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A STUDY OF LAMENESS IN DAIRY COWS WITH REFERENCE TO NUTRITION AND HOOF SHAPE

by

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CONTENTS

SUMMARY

1. The literature review covers the incidence of lameness in dairy cows and the predisposing management factors associated with lameness. Housing in relation to lameness and the nutritional causes of lameness, with particular reference to laminitis, are discussed. The genetic basis of lameness and the relationships between locomotion and hoof trimming are also reviewed. An outline of hoof structure, function and formation is given to enable a better understanding of the processes involved in hoof lameness.

2. In Experiment 1 two groups of 24 cows were offered 2 levels of concentrate from out-of-parlour feeders (7 kg/day versus 11 kg/day), plus silage ad libitum, during weeks 3 to 22 of lactation. Locomotion scores and rates of clinical incidence of lameness indicated that the high level of concentrate increased lameness. However, there were no significant effects on hoof shape.

3. In Experiment 2 the effects of protein level (161 g/kg crude protein versus 198 g/kg crude protein) and Dutch hoof trimming (trimmed versus untrimmed) on four groups of 12 cows were examined, during weeks 3 to 26 of lactation. Metabolisable energy intakes and the concentrate to silage ratios were maintained at the same level for all four treatments. Trimming and the high protein level significantly increased locomotion score and clinical incidence of lameness. Trimming also increased hoof growth and reduced heel bulb hardness. No relationships between lameness and various blood parameters were found. There were indications that lame cows did not perform as well as those who were not lame.

4. In Experiment 3 the effects of concentrate to silage ratio (38:62 versus 63:37) and Dutch hoof trimming (trimmed versus untrimmed) on four groups of 12 cows were investigated, during weeks 3 to 26 of lactation. The high concentrate to silage ratio significantly increased locomotion score and trimming reduced locomotion score although not significantly. Higher incidences of clinical lameness in the high concentrate to silage ratio and

untrimmed treatments were found. The high concentrate to silage ratio significantly reduced the hardness of the abaxial wall and sole, and as in Experiment 2 trimming increased hoof growth. Again there were indications that poor locomotion and lameness were associated with losses in production.

5. Behaviour was observed during three 24 hour periods in each of the three feeding trials. Negative correlations between time spent feeding and positive correlations between lying time and locomotion score were found. The number of social interactions decreased as locomotion score increased.

6. These experiments indicated that high levels of concentrate and protein, and high concentrate to silage ratios resulted in poorer locomotion and higher incidences of clinical lameness. Trimming appeared to improve locomotion, although its benefits in correcting hoof shape and weight distribution were not long term due to its effect of increasing hoof growth. Hoof shape and hoof hardness were also related to locomotion and lameness.

ABBREVIATIONS

cm	Centimetres
CP	Crude Protein
D, D value, DOMD	Digestibility of the Organic Matter in the Dry Matter
DM	Dry Matter
EAAP	European Association of Animal Production
g	Gram
kg	Kilogram
MAFF	Ministry of Agriculture, Fisheries and Food
ME	Metabolisable Energy
MJ	Megajoule
N	Nitrogen
NS	Not Significant
o	Degrees
RDP	Rumen Degradable Protein
SED	Standard Error of the Difference
UDP	Undegradable Protein
*	$p<0.05$
**	$p<0.01$
***	$p<0.001$

INTRODUCTION

Lameness in dairy cows is important not only for its animal welfare implications, but also because of any potential economic loss. Any restriction of a cows movement, and therefore a reduction in time spent standing and walking, may indirectly affect milk production and reproductive performance. Thus, a knowledge of the way in which lameness alters the behaviour pattern of a cow would be informative.

Lameness is a condition resulting from a number of different lesions or diseases affecting the foot or limb. Lameness may be caused by several predisposing management and hereditary factors, which may act singly or together. Identification of the causal factors and the way in which they inter-act is difficult, and it is therefore not unexpected that few large scale experiments have been carried out to elucidate which factors, and in particular nutritional factors, are of importance.

An understanding of hoof structure and function is important, since much of hoof lameness is thought to result from alterations in hoof shape and hardness. Therefore, it would be useful to know why and how the various enviromental factors bring about such alterations.

INCIDENCE

The economic loss caused by lameness has been estimated at #35 million per annum (Kelly, 1981), through veterinary treatment, losses in milk yield, reduced fertility and culling. Several surveys have been undertaken to estimate the incidence of lameness in Britain. However, strict comparisons between such surveys can not be made, since data collection methods and diagnostic criteria differ. Reported levels of incidence will also vary widely depending on whether the incidence is based on diagnoses made by the farmer, or on veterinary records. Incidence figures taken from veterinary records are probably considerable underestimates of actual levels of lameness, since many lame cows are treated by the farmer with no referral to the veterinary practice.

Most lameness has been associated with the hoof. A survey

of veterinary records kept on seven Cheshire farms showed that of the lameness cases, only 8% involved leg lesions whereas 92% involved hoof lesions, of which 30% occured in the interdigital area and 70% in the claws (Prentice and Neal, 1972). Lesions were also found to be significantly associated with the hind hooves and with the lateral claws. In a major survey in 1977, which used veterinary records and involved 3% of the British dairy, an average incidence of 5.5% was found; 88% of lesions occurred in the claws (Russell et al, 1982). Eighty-four per cent of these were found in the hind feet with 85% being found in the lateral claws. The commonest lesions were foul in the foot (16.7%), solar ulcer (13.6%), punctured sole (10.4%) and foot rot (8.7%). The remaining 12% of lesions occurred in the hind limbs. Trauma was the main cause of leg lesions, which were most frequently observed in the joints and ligaments.

Lameness has been thought to be more prevalent in areas of of high rainfall (Eddy and Scott, 1980). Similarly, a study of lameness in Ireland showed seasonal variations in incidence which may have been associated with rainfall (Arkins, 1981). A seasonal effect was also found by Rowlands et al (1983), who found that the incidence rate was lower in summer (0.71 cases per 100 cows per month) than in winter (0.87 cases per 100 per month).

Stage of lactation and age have also been related to lameness. Rowlands et al (1985) reported a higher level of lameness in the first month of lactation (15% of all cases), and an increasing susceptibility to lameness with age. Ten year old cows were over four times as likely to develop lameness as three year old cows; this was due to an increased incidence with age of white line abscess and solar ulcer.

HOOF STRUCTURE AND FUNCTION AND HORN FORMATION

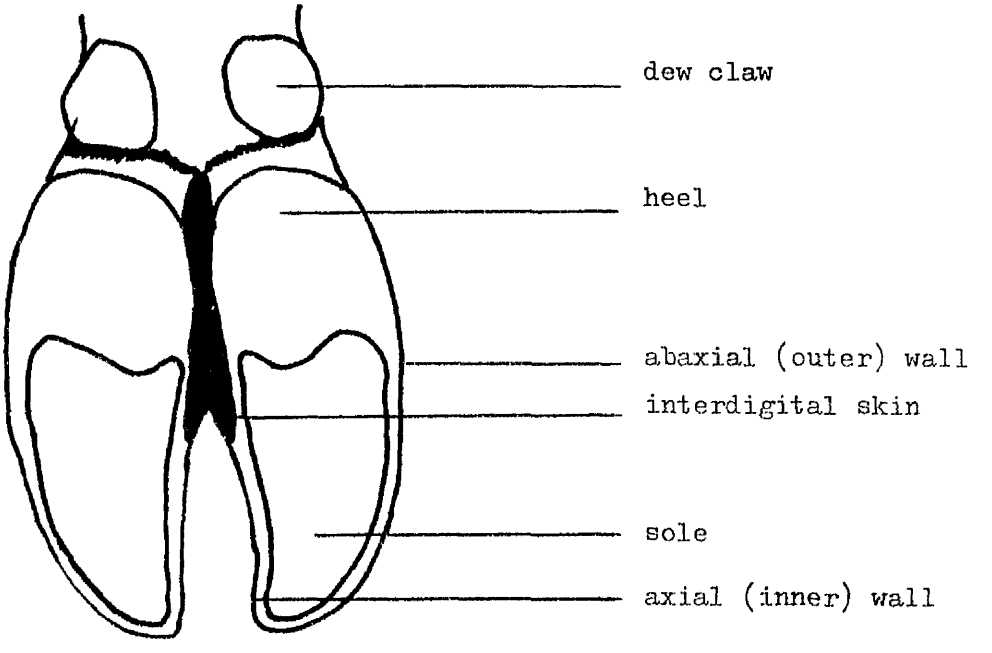
A cows hoof consists of two large claws or digits, separated in their lower part by the interdigital space. The claws have a bony central core overlaid by soft tissues and have an outer

surface of horn. For a diagram of hoof structure see Figure 1.1.

The coronet is the border joining the skin and hoof horn, distal to which is the periople. This periople is a layer of soft horn which reaches down distally from the coronet for about 1.5 cms. The white line composed of soft horn tissue, non-pigmented tubular and inter- laminar horn joins the rigid wall with the non-rigid sole. The sole is composed of tubular and inter-tubular horn, and the horn is thicker along the outer border than on the inner border. The sole is normally concave, so the weight is mainly borne by the heels and walls, although some may be taken by the outer borders of the sole. If the hooves become excessively worn, the sole flattens, making it susceptible to trauma and contusion. The sole is joined to the heel as a small depression, the heel-sole junction, which is vulnerable to injury. The horn of the heel contains incompletely keratinised tissue and more inter-tubular horn than that of the walls or sole and is thicker and softer. Between the dermis of the heel and the deep flexor tendon of the hoof is the digital cushion. This cushion consists of fibroelastic and adipose tissue and functions as a shock absorber, flattening and spreading laterally towards the walls when the hoof makes contact with the ground.

The functional anatomy of the ligaments, tendons, bones and joints is described in detail by Greenough et al (1981). The pedal bone is the most important of these structures, because of its relationship with the corium. The corium (dermis) is a layer of tissue supplied with nerves and blood vessels, which furnishes nutrition to the hoof. The pedal bone both rests on and is suspended in the corium within the hoof (see Figure 1.2). Through the influence of body weight the corium is under pressure between the pedal bone and the horny sole of the weight bearing claw. This aspect is fundamental in understanding hoof lameness and the reasons for trimming. The relative position of the pedal bone to the corium varies in different parts of the hoof. In the anterior part of the sole the corium is adjacent to the pedal bone, and therefore immovably fixed in the horny tissue, whereas under the heel and the rest of the sole the pedal bone is loose due to the presence of the digital cushion. Thus the weight of

Figure 1.1 Hoof structure



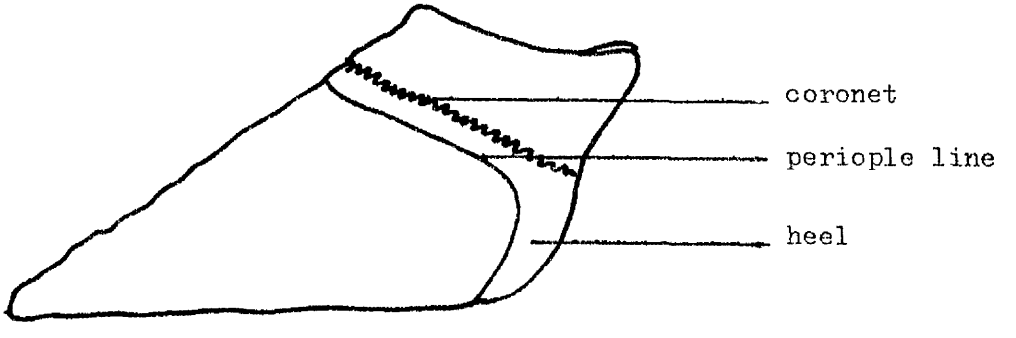
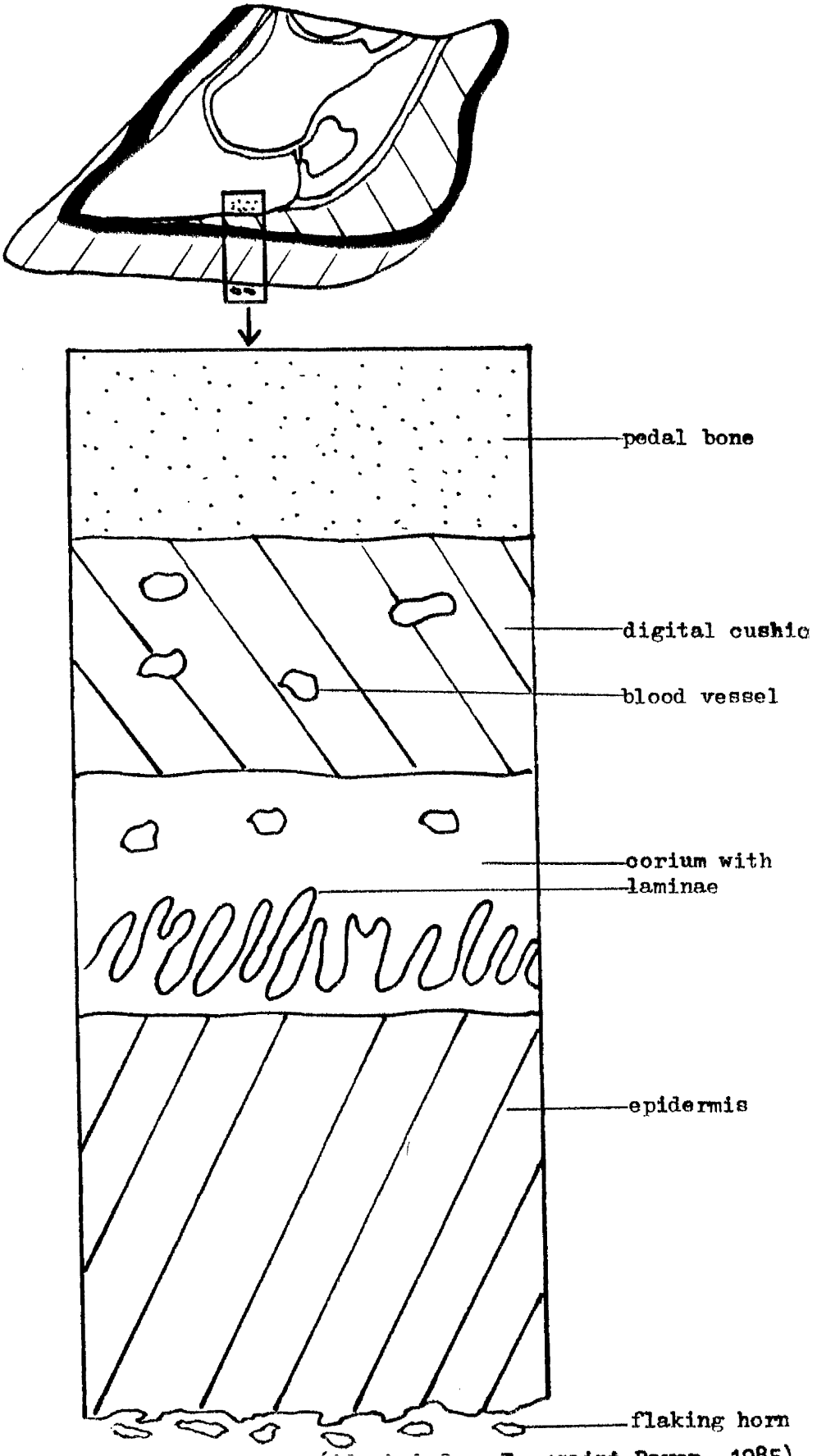


Figure 1.2 Diagram to show the relationships between the pedal bone, digital cushion, corium and epidermis.



the cow will cause the pedal bone to sag axially and backwards. On the axial side in the posterior part there is a thickening of the pedal bone called the axial prominence. Thus under loading the corium in this region will be slightly compressed. Under normal conditions such compression will not cause foot problems, but if there is prolonged overloading especially when coupled with some corial disorder, this compression may lead to the typical solar lesion frequently found in this region (Toussaint Raven, 1985).

The corium (modified skin dermis) underlies the epidermis, as shown in Figure 1.2, and via its blood vessels supplies nutrients for horn formation. Horn formation occurs in the stratum germinativum, the deepest layer of the epidermis, which is closely associated with the corium. Cell division takes place in this layer, nutrients diffusing through the basement membrane of the corium. The newly produced cells, containing precursors of onychogenic (horn producing) substances, push the previously formed cells towards the epidermal surface. As the cells move they form the horny substance at their surface in a process called keratinisation.

Keratins are the main chemical constituent of horny tissue. They are a large group of proteins, which have a variable amino acid composition and contain disulphide linkages. The disulphide bonds are formed by the oxidation of sulphydryl groups of residues of adjacent polypeptide chains (Matoltsy,1958). Many of these residues are cystine residues. Sulphur containing amino acids as a whole constitute 7.3% of the total protein of the hoof wall and 5.7% of the sole (Maclean, 1971a). The disulphide bond is an important factor in the healthy functioning of the hoof since it is responsible for the high mechanical resistance of keratin and also has a role in determining the elasticity of the horn tissue.

HOOF MEASUREMENTS

Hoof horn growth

The hoof horn grows through cell division in the stratum germinativum of the epidermis and by subsequent keratinization. Wearing occurs at the bearing surface due to the force of traction and the spreading of the toe during walking (Fessl, 1974).

Rates of horn growth vary between and within hooves. There is a difference in shape between the outer and inner claws of the hind foot, which is perceptible from the age of 18 to 24 months. The outer claw is larger than the inner claw with a flatter sole, a wider bulb and a higher heel (Toussaint Raven, 1973). Prentice (1973) found that the posterior wall regions grew on average 40% faster than the toe regions which confirmed Greenough's (1962) observations about the differential growth rates within the claws. However, the latter observations were non-quantitative. Hahn (1979) and Simon and Leeman (1965) also found that the lateral wall grew faster than the dorsal wall. Rear hooves were found to grow faster than front hooves both in cattle and ponies (Simon and Leeman, 1965; Butler and Hintz, 1977; Hahn 1979). In contrast, Prentice (1973) reported that the claws of the fore feet grew faster than those of the hind feet; this was attributed to the greater weight borne by the fore feet. Although excessive and unequal weight distribution may be related to growth rates in over-grown claws (Toussaint Raven, 1972; 1973), growth rates are more likely to be determined by nutritional factors and the health of the horn producing tissues.

Nutrition is the most important factor influencing horn growth. Whilst adequate amounts of dietary protein and carbohydrate are required to form high quality horn, it is the type and concentration of amino acids, in particular the sulphur amino acids, which is the main determinant of horn growth rates. Although little is known about the relationship between amino acid supply and horn growth, the effects of amino acid levels on wool growth, which is analagous to hoof growth, have been well documented.

Abomasal infusions or intra-peritoneal injections of

methionine, methionine hydroxy analog and cysteine have stimulated wool growth in sheep (Reis, 1967; Wright, 1969). Optimal levels for each amino acid exist, with adverse effects occurring at higher than optimal levels due to amino acid imbalance, toxicity or through effects on amino acid transport (Reis and Schinckel, 1964; Reis, 1967). Methionine may have a specific stimulatory effect on wool growth through its role in amino acid transport (Christensen, 1963), or as an initiator in protein synthesis (Noll, 1966), in addition to being a source of cysteine (Reis, 1967).

Stage of lactation has been associated with hoof growth by Bemis et al (1985), who reported that hooves grew slower in early lactation than in late lactation. However, Hahn (1979) showed that although there was a stage of lactation effect on the wear rate of front feet, there was no such effect on hoof growth. Similarly, Clark and Rakes (1982) found that number of days post-partum were not significantly related to hoof growth, but this stage of lactation effect may have been confounded by photoperiod or ambient temperature effects.

Levels of milk production may also affect hoof growth. Dietz and Koch (1972) found that high producing cows had the lowest rates of growth among several herds of cows. Milk producing tissues may compete with horn producing tissues for protein components. Wool growth and milk production have also been associated. Oddy (1985) showed that wool growth deficit was directly proportional to milk production, and Corbett and Furnival (1976) suggested that wool growth depression during lactation may have been related to milk production levels.

Hormonal levels may influence hoof growth. Depression of wool growth is associated with hormonal effects on the partition of ingested nutrients, and although increases in voluntary food intake occur during lactation, wool growth may be reduced by 20% to 40% in late lactation (Corbett, 1979). The release of a hormone in response to photoperiod duration, may also augment keratin synthesis (Clark and Rakes, 1982). Photoperiod in addition to ambient temperature may also explain seasonal trends in hoof growth. Hahn (1979) found that greatest growth occurred in late spring and early summer, and that lowest growth occurred in winter. Cyclical changes in hair growth are also thought to be

due to photoperiod changes (Yeates, 1955; Peters et al 1976). Increasing rates of growth with increasing ambient temperature have been reported (Bemis et al, 1985), and these increases may be due to an increased blood flow through the skin or to a speeding up of temperature dependant biochemical reactions (Bottomley, 1979).

Amino acid levels may be of particular importance when high rates of horn growth are being determined by other factors, such as those outlined above. However, no firm conclusions can be drawn from the literature, since interactions between these factors has not been investigated.

Hoof hardness

The hardness or resistance of horn to penetratation is partly dependant on its moisture content (Fritsch, 1966). Hydrated horn was found to be considerably softer than dry horn (Prentice, 1970), with the lowest readings being obtained from hydrated mid-sole horn. This region corresponds to the area most frequently associated with solar penetrations and ulcers.

Nutrition, in particular levels of sulphur amino acids, is also important in determining hardness. Softer keratin tissue has been attributed to decreased disulphide bonding and lower levels of cysteine in the keratin protein (Clark and Rakes, 1982).

Work at the Institute for Research on Animal Diseases (1983) has shown that as horn hardness increses, water, potassium and total ash contents decrease. The claw horn of animals with solar ulcers was also found to be softer, the softness being associated with increased water, magnesium and copper and decreased zinc contents. Whether they have a role in the etiology of claw disease or are merely secondary effects remains uncertain.

Hoof shape

The length of the dorsal wall is important, since it determines the weight distribution within the claw, and it is also associated with the efficiency of the wall to perform its function of transmitting pressure to the skeletal structure (Hahn, 1979). Toe length is dependant on the relative rates of hoof growth and wear.

The angle of toe measures the inclination of the tubuli, which are involved in the absorption of pressure. It has been found that angles decreased and lengths increased as cows aged

(Hahn et al, 1978).

Heel depth is subject to growth and wear, and infections such as those caused by Fusiformis nodosus, which result in horn erosion. The depth and integrity of the heel tissue determine its ability to act as a shock absorber.

Other hoof shape parameters include the area of the bottom of the hooves, which is larger for the front hooves than for the rear ones (Fessl, 1968). Similar results were found by Meyer et al (1968). Despite the larger area of the front claws, Distl et al (1984) found that there was a greater pressure on the ground surface of the front claws than on the rear claws. It has been estimated that the fore legs of cattle carry about 55% of total body mass rising to about 75% during slow movement (Irps, 1983), and Greenough et al (1981) have suggested that the hind legs are used more for propulsion.

Hoof strength and hoof elasticity

Hoof strength is a measure of the force required to produce tissue failure, (Webb et al, 1984), and the levels of disulphide bonding in the keratin have been related to this variable (Mercer, 1961). The density of horn tubules is also important, the more horn tubules per unit of horn surface, the stronger the horn (Mauske, 1972). Hoof elasticity is a measure of how far a tissue deforms under a given load (Webb et al, 1984). A more elastic hoof is thought to be more resistant to the abrasive wear of concrete, because of its ability to expand and contract (Clark and Rakes, 1982). Such resistance may be lowered by progressive dehydration; results have indicated that as the moisture content of the horn increases, the elasticity decreases (Prentice, 1970).

Thus, several factors, such as levels of sulphur amino acids and moisture content, through their effects on various measurements, determine the overall quality of hoof tissue, and therefore the chances of whether hoof lameness develops or not. Additionally, the molecular structures contributing to the different properties may be as different as their mechanical functions require. For example, whereas the hardness of the wall

is largely determined by levels of keratin, it is likely that the underlying connective tissue also contributes to the relative softness of the heel bulb (Webb et al, 1984).

The relative importance of each factor in influencing the various hoof properties, and the nature of any interactions which may occur between such factors, needs to be more clearly elucidated before an improved understanding can be reached of how and why hoof tissue malfunctions.

TYPES OF LAMENESS CAUSED BY HOOF PROBLEMS

Lameness has been defined as the clinical sign of disease or abnormality of the musculo-skeletal system, and has been classified in terms of the character of the presenting symptom (Greenough et al, 1981). It has been more generally defined as any restriction of the voluntary movement of the animal (Hahn, 1979).

Much of the confusion surrounding the classification of the diseases of the hooves and limbs is due to the lack of recognition of the frequent occurrence of lameness as a symptom of some multi-factorial disease syndrome. Further problems arise since more than one type of lameness may be present concomitantly. However, nomenclature and definitions of diseases of the hoof were agreed upon by a group of veterinarians with the support of the International Council of Disorders of the Ruminant Digit at Skara in 1978, and outlined by Weaver et al (1981). These diseases are described in Appendix 1, and are listed below.

Pododermatitis aseptica diffusa (laminitis)
Pododermatitis circumscripta (solar bruising/ulcer)
Dermatitis interdigitalis @
Phlegmona interdigitalis (foul-of-the-foot)
Pododermatitis septica (solar penetration)

Hyperplasia interdigitalis (inter-digital growth)
White zone disease (white line disease)

@ foot rot is usually described as Dermatitis interdigitalis together with underrunning

THE GENETIC BASIS OF LAMENESS

Although several workers have investigated the heritability values resulting from various subjective evaluations made on the feet and legs of cattle, research on the genetic variation of foot disorders is scarce. Systematic measurements of hoof hardness, growth, wear, elasticity and strength, or the components of these characteristics, which are likely to be underlying causes of lameness, have been little examined.

Peterse and Antonisse (1981) found that differences in the incidence of solar lesions between progeny groups, which could be detected at an early stage of first lactation, were sufficiently significant for selection of cattle with lower incidences of solar lesions. However, there were no indications that interdigital dermatitis could be diminished by selection. Some of the genetic aspects of solar ulcers and interdigital dermatitis in relation to claw measurements were studied by Smit and Verbeek (1984). Genetic correlations between toe length, toe angle and heel depth with solar ulcers were greater than with interdigigtal dermatitis. Greenough et al (1981) have also suggested that inheritance factors involved in the absorption of concussion, for example, too straight hocks or upright pasterns, predispose to solar ulcers. Too straight or too angulated limbs are also thought to to predispose to arthritis (Amstutz, 1965). A genetic predisposition to laminitis has been proposed by several authors (Steele-Bodger, 1960; Nilsson, 1963). Mead et al (1949) indicated that a single autosomal recessive gene in Jersey cattle causes animals to develop laminitis. Interdigital growths (Hamori et al, 1963), corkscrew claws (Greenough et al, 1981) and hoof overgrowths (Glicken and Kendrick, 1977) are also thought to have an important genetic component.

Several workers have examined the heritability of leg and

foot conformation with conflicting results. These discrepancies are probably due to the subjective nature of the evaluation of conformation, to differences in statistical models used and to biases in scoring. For example, Distl et al (1984) found that interactions between sire and age in Simmental bulls did not allow an unbiased estimation of heritability for various hoof measurements. Additionally, not all heritability values have taken into account age, stage of lactation, housing and feeding systems. Norman and van Vleck (1972) observed that differences in age and stage of lactation were apparent for many traits in Holstein cows, although interactions between age and stage of of lactation were relatively small. Hahn et al (1984) found that the the stage of lactation effect was highly significant in the first lactation, less in the second lactation and nil in the third and later lactations. Although sire effects were significant for most hoof shape measurements (Hahn et al, 1978; Hahn et al, 1984), there were also herd effects (Hahn et al, 1984). Generally, low heritabilities for leg and foot conformation have been obtained (O'Bleness et al, 1960; Cassell et al, 1973; Hay et al, 1983), indicating that enviromental differences, for example, housing and nutrition, have such a significant effect on scores that genetic differences, should they exist, are masked (Rennie et al, 1974).

Other hoof parameters examined include hoof growth and wear rates, and tubular tissue measures. An international experiment involving ten strains of Friesian cattle from ten different countries has shown that growth and wear rates are significantly different in the various sub-groups (Drodz, 1980). Distl et al (1981) also found that genetic differences existed between progeny groups for electrophoretical measurements of soluble proteins, which were significantly correlated to rate of hoof wear and water content of the claw. Genetic differences were also observed for number, diameter and area of tubes of hoof horn. Amounts of tubular tissue relative to inter-tubular tissue are thought to be related to hoof strength.

Although there is some evidence of a genetic effect on lameness and various hoof parameters, lameness problems are

better explained by a complex interaction of several factors including breeding, nutrition and housing.

NUTRITIONAL CAUSES OF LAMENESS WITH SPECIAL REFERENCE TO LAMINITIS

Nutritional disorders have been implicated as important etiological factors in the development of laminitis and with associated hoof problems, for example solar lesions. However, even though comprehensive descriptions of the pathology of laminitis exist (Nilsson, 1963; Maclean, 1971; Andersson and Bergman, 1980), the actions of the nutritional factors at a cellular, biochemical and physiological level are little understood.

Consumption of large quantities of concentrates or succulent forages have been associated with the onset of laminitis (Morrow, 1966) as have changes in feeding regime, especially around parturition. Peterse and van Vuuren (1984) examined the effects of rate of concentrate increase on the incidence of foot lesions in freshly calved heifers. In one group there was a change from 1 kg of concentrates and ad lib silage pre-calving, to 10 kg concentrates and ad lib silage post-calving, over seven days. The second group was fed the same ration, but with the change occurring over a period of sixteen days. A similar incidence and severity of solar lesions, which were considered to be the symptoms of laminitis, were found in both groups. The absence of a significant effect may have been due to the relatively small difference between the groups in the rate of concentrate increase. However, the lack of a significant difference in frequency of such lesions, is not necessarily indicative of the absence of laminitis, since laminitis may have been present sub-clinically or its symptoms may have taken another form. The results of Peterse and van Vuuren (1984) are in contrast to those of Trimberger et al (1972), who studied the effects of liberal grain feeding over a three year trial period. During the first year of the trial, but not during succeeding lactations, symptoms of acidosis and laminitis were observed and these were attributed to the rapid change over from a forage based diet to a grain based diet.

Low fibre, high starch diets have also been linked with

laminitis, and it is likely that a metabolic disorder such as acidosis is involved. Livesey and Fleming (1984) investigated the effects of early lactation diets with different crude fibre contents, but with similar energy intakes on laminitis and solar ulcers. Different early lactation diets were imposed on three pre-calving diets, two of which would have allowed the rumen to adapt to starchy substances before calving. Although the cows on the low fibre ration had significantly higher incidences of laminitis and solar ulcers than those on the high fibre ration, pre-calving diets did not appear to affect the results. This was attributed to the substance responsible for precipitating laminitis, being a product produced in proportion to the level of starch in the diet, rather than to a product produced transiently while the rumen microflora adapted to the new diet. However, the trial period may have been too short to allow for a true assessment of the incidence of solar ulcers.

In another study, Peterse (1979) compared the incidence of solar lesions in two groups of dairy cattle fed different levels of concentrate. The group fed 6 kg concentrates before calving had a significantly higher incidence of solar lesions after calving, than those fed 1 kg of concentrate.

Thus, whilst the results from the trials of Peterse (1979) and Peterse and van Vuuren (1984) suggest that level of concentrate fed may be of more importance, the results of Trimberger et al (1972) show that change in diet has a greater influence on the development of lameness. Further trials are needed to establish which factor is of more importance, and whether any interactions occur between rate of change and level of concentrate fed.

Lactic acidosis caused by excessive ingestion of feeds rich in readily available carbohydrates has frequently been associated with the etiology of laminitis. Weaver (1979) found that administration of lactic acid to lambs brought about acute laminitis within 24 hours. The ingestion of starchy feeds is followed by a marked change in rumen microflora; there is an

increase in Streptococcus bovis, which ferment dietary carbohydrate to produce large quantities of lactic acid. When the pH falls below 5, lactic acid will be absorbed from the rumen into the bloodstream. A fall in blood pressure, due to blood dehydration, leads to a sluggish circulation, which in turn causes a reduction in the nutrient and oxygen supply to the corium. Endotoxins associated with acidosis are thought to have a toxic influence on the capillary walls, which also causes an insufficient supply of nutrients to the keratin forming cells with subsequent synthesis of structurally incompetent keratin (Andersson and Bergman, 1980).

Faulty keratinization also occurs in laminitis, but whether this is due to stagnation of the blood, or to a loss in capillary integrity, or to a combination of both is uncertain. Whether the factors involved in laminitits and acidosis are similar, or whether it is the acidosis itself which predisposes to laminitis is also unclear. Certainly the biochemical and pathophysiological relationships between acidosis and laminitis remain unresolved.

Evidence for the existence of a relationship between protein level or protein source with lameness is scarce. However, some work has suggested that a protein related allergic-histaminotic phenomenon may be involved in the etiology of laminitis (Nilsson, 1963), and Bazeley and Pinsent (1984) have related the feeding of a high protein supplement and a high percentage of free ammonia in silage to a high incidence of acute laminitis.

Nilsson (1963) found that protein rich concentrates led to allergic reactions associated with laminitis, and in subsequent work Nilsson (1966) suggested that the thromboses found in laminitic cows may have developed as allergic-histaminotic phenomena. This agrees with the work of Jimenez-Dial (1959), who proposed that a connection between the occurrence of thromboses and allergic manifestations existed. Similarly, Morrow (1966) suggested that the breakdown of protein in the digestive tract, which resulted in the release of large amounts of histamine and histamine intoxication, led to an enlargement of the vascular

laminae of the hoof.

Histamine has also been implicated by other authors as an etiological factor in the pathogenesis of laminitis. The histopathology of laminitis was thought to resemble the circulatory effects of histamine capillary permeability and arteriolar dilatation (Brent, 1976), and anti-histamine therapy was found to produce favourable results if given in the early stages of laminitis (Nilsson, 1963; Jubb and Kennedy, 1970; Chew, 1972). It has also been suggested that a release of histamine is involved in the inflammatory response of laminitis (Akerblom, 1963; Nilsson, 1963; Maclean, 1966). Maclean (1970) also found that serum histamine was slightly elevated during acute laminitis, and became further elevated as laminitis progressed to a chronic stage. Previously, Maclean (1965) observed that high blood histamine levels were present shortly before the onset of other symptoms of laminitis. However, the origin of such elevated blood levels is unclear, since serum histamine may come from the laminitic tissue itself, from endotoxins which are released when rumen bacteria lyse at low pHs (Brent, 1976), or from dietary sources of histamine, such as grass silage (Sjaastad and Stormorken, 1963).

The role of ruminal histamine, whether from lysing bacteria or silage, in predisposing to laminitis is unlikely to be important, since histamine appears to be readily excreted in the urine without any apparent harmful effects. Sjaastad and Stormorken (1963) found large increases in the excretion of acetyl-histamine in the urine, with only a minimal increment in free histamine. Additionally, absorption of histamine across the rumem wall into the blood stream was thought to be unlikely. Orally administered histamine was not found to influence the physiological condition of the rumen, and this was attributed to poor absorption of histamine through the rumen wall epithelia. However, histamine may be absorbed during parturition, since parturition may supply by some mechanism the necessary stimulus to allow absorption from the alimentary tract (Maclean, 1965). This may correspond to the finding that acute laminitis frequently

occurs soon after calving (Greenough et al, 1981). Nilsson (1966) suggested that damage to the mucosae of the abomasal wall, which is associated with catarrhs found in some laminitic cows, would allow the entry of histamine into the blood stream.

Thus, until the the possible pathways taken by histamine from the rumen to the capillaries in the corium are clarified, and until the portion of the serum histamine level which is attributable to histamine originating from the rumen can be determined, the role of histamine in the etiology of laminitis will remain uncertain.

Other authors have suggested that toxins of proteinaceous origin, rather than histamine, are important in the predisposition to laminitis (Urmas,1968; Chew, 1972). Such toxins are thought to account for the drain on the chondroitin sulphates of horn tissue, which leads to deranged epidermal horn formation (Urmas, 1968). It is likely that the actions of such toxins are similar to those which originate in the acidotic rumen.

Protein and in particular sulphur amino acids, have also been related to hoof growth, as discussed on page 13, and overgrowths resulting from high growth rates can be a major cause of lameness (Toussaint-Raven, 1972; 1973). Sulphur amino acids are also important with respect to hoof hardness. Poor quality horn production may lead to insufficient protection of the underlying tissues (Greenough, 1962; Smedegaard, 1964).

The relative importance of energy level, protein level, energy source, protein source and change in diet, in relation to the etiology of laminitis, needs further clarification. The possibility of interactions or synergistic relationships occurring between the various dietary components also requires examination, since it appears that toxins of both acidotic and proteinaceous origin are important factors in the development of laminitis. Additionally, before a better understanding of the role of nutrition in lameness can be reached, adequate assessments and diagnoses of lameness need to be

made. Thus, the role of dietary factors in predisposing to lameness remains uncertain.

HOUSING AND LAMENESS

There have been many developments in housing systems in recent years, but few designs have been primarily concerned with combating lameness. Few criteria for an optimal environment (climatic, structural and social) for the cow have been developed, since there are no standardised, systematic methods for describing cattle housing, nor is there an in-depth understanding of the effects of various housing factors, for example, flooring and cubicle dimensions, on foot and leg health. Compounding these problems is the multi-factorial nature of lameness making it difficult to assess the importance of housing in relation to other etiological factors.

Several surveys have been undertaken to establish the types of housing systems, which are least predisposing to lameness. However, there is difficulty in comparing surveys, since there are considerable variations between surveys in levels of moisture and slurry, nutrition and exercise.

An extensive survey in Sweden (Ekesbo, 1966) was undertaken to compare incidences of lameness in loose housing and tied stalls. No relationship between housing type and the incidence of foul-of-the-foot or foot rot was found, although foul-of-the-foot was associated with husbandry. In loose housed herds the incidence was higher where the lying and feeding areas were churned up and dirty, than in herds with clean areas. However, Grommers (1968) found that the incidence of foul-of-the-foot was significantly ($p<0.01$) higher with loose housing than with tie stalls, but this may have been attributable to the longer distances that the loose housed cows had to walk on muddy paths. Solar ulcers may be associated with enviroments with hard floors (cowsheds and cubicles with concrete yards), which are more likely to cause bruising of the junction between the sole and heel than straw yards (Rowlands et al, 1983). White line disease was

found to be more prevalent in straw yards and cubicles than in cowsheds; in contrast aseptic laminitis was more common in cowsheds than cubicles (Rowlands et al, 1983).

Adequate levels of bedding were observed to reduce the incidence of traumatic leg and hoof injuries (Ekesbo, 1966), probably through their preventing excessive solar abrasion and exposure to a hard floor, which would cause tissue damage to the leg with subsequent oedema formation. Maton and Moor (1975) found that there were twice as many claw injuries in cubicle houses as compared to in straw yards, and leg injuries were observed to be higher with tie stalls as compared to loose housing (Grommers, 1968; Rowlands et al, 1983; Seibert and Senft, 1984).

The presence or absence of slats may be more important in determining the incidences of traumatic hoof injuries and hoof problems caused by disease-producing organisms than housing type. Although solid floors in loose housing are generally regarded as predisposing to more disease than slatted floors (Minguy, 1974; Blom, 1982 b; Junge and Ernst, 1983), slats may lead to fractured claws, hock hygroma, sprains and torn muscles (Dietz and Koch, 1972). A study of the foot conditions of calves and young cattle kept on slatted floors also revealed quality changes in the horn of the sole and claw loading, which were causative factors in claw malformations (Schmoldt and Heyden, 1973).

Foot-floor interactions are important in the understanding of the relationship between housing and lameness caused by hoof problems. Webb and Nilsson (1982) have specified four floor properties, abrasiveness, hardness, friction and surface profile. However, these properties are only applicable to the floor under specific conditions, since the properties of the floor are altered by changes in moisture and slurry levels. Experiments have shown that with the same load, claw abrasion on a wet floor is 83% greater than on a dry floor (Gravert, 1977).

Levels of claw abrasion are important, since over-abrasion predisposes the cow to solar contusions and entry of infection , whilst inadequate levels of abrasion result in overgrowths. Thus

an equilibrium between hoof growth and wear needs to be maintained to prevent hoof problems occurring. Such an equilibrium will be effected by the abrasiveness of the floor, hoof hardness and elasticity, amount of exercise and dietary and genetic factors which determine hoof growth and wear. Lasczka (1965) suggested that 30 minutes exercise a day on hard ground would prevent overgrowths in bulls, and Chwojnowski (1965) found that exercise was an important factor not only in preventing lameness, but also in curing lameness.

The surface profile and the hardness of a floor may influence lameness through the stresses and strains that they produce in the hoof. External, mechanical stress applied to the hoof will lead to physical injury, when it is greater than the strength of the hoof tissue (Webb and Nilsson, 1982). Shock et al (1981) showed that higher stresses are set up in pigs hooves when the floor is highly profiled, and Baggott (1982) suggested that vertical cracking from the coronet may be caused by sudden overloading of the cows hoof on uneven surfaces. Floor hardness determines the maximum stress that a tissue receives. By deforming, a floor reduces the contact pressure on a limb by redistributing the load over a wider area, thus reducing the mechanical stress (Webb and Nilsson, 1982).

Friction determines the conditions under which slipping occurs. Coefficients of friction decrease when floors are covered with slurry or water. Nygaard (1979) suggested that the use of bedding with rubber mats was imperative in the prevention of traumatic injuries, since without bedding mats tended to become wet and slippery. Gjestang and Loken (1980) showed that there was a rapid decrease in slipping and therefore injury, when coefficients of friction were increased.

Slurry and mud influence lameness through their effects on floor properties, and by providing an environment for the multiplication of Fusobacterium necrophorum and Bacteroides nodosus, which are important etiological factors in the development of foul-of-the-foot and foot rot, respectively. Dried

mud may also bruise the interdigital skin sufficiently to lower its resistance to Fusobacterium necrophorum, and pieces of gravel underfoot may result in contusion and impaction of the sole resulting in solar ulceration (Chew, 1972).

Sufficient bedding is important in reducing limb bruising and abrasions, which are caused by trauma. Acceptability of the bedding to the cow should be maximised, so that the cows feet spend a maximum amount of time on its relatively hygeinic and non-erosive surface. Choice tests have shown that cubicles with soft floors are occupied more than those with hard floors (Irps, 1981). Sawdust thickness is important; occupation time was found to increase with increasing thickness up to 15cm and then decrease (Wander, 1974). Foothold insecurity may have accounted for the decrease (Lasson and Boxberger, 1978). However, Kovalcik et al (1982) showed that there was no overall preference between cubicles with straw and cubicles with sawdust.

Lameness which is related to housing factors could be reduced if a more zoocentric approach, which took account of the cows physical requirements was adopted in the design of cattle housing. Further understanding of the relationships between housing factors, for example, floor type, bedding and area available for exercise, and biomechanical aspects of the hoof, would thus aid the development of more appropriate housing designs.

LAMENESS AND PRODUCTION

Although the importance of lameness in terms of production losses has been emphasized, few studies have quantified or allocated such losses. There are difficulties in estimating the losses annually incurred by the British dairy industry, since surveys on the incidence of lameness itself have been based on different reporting procedures; some surveys have used data based on farm records whilst others have used veterinary records.

In a study of four herds in New Zealand the behaviour and

production of lame cows were compared with their non lame counterparts. The lame cows were disinclined to walk, spent long periods recumbent, seldom sought food and lost body weight very rapidly. Lameness was also directly linked to poorer milk yields. Although initial losses in milk production incurred during lameness appeared to be of minor importance, losses for the full lactation were cumulative and substantial, and the higher the incidence the greater was the loss (Dewes, 1978). Long term effects of lameness on production have also been found by Dorynek et al (1980) who examined 68 pairs of lame and sound cows. The 305 day yields of the lame cows were significantly ($p<0.01$) lower than those of the sound cows.

The losses in condition and milk yield may be due to the effects of lameness on altering the cow's behaviour pattern. It is likely that lame cows spend more time lying and therefore less time feeding than non lame cows. In a trial involving 60 cows housed in loose- housing yards, it was observed that when cows became ill they fed less frequently and for shorter periods, and in the few cases of clinical lameness reductions in food intake seemed to occur before clinical symptoms were observed (Collis et al, 1980). However, the extent to which lameness prevents feeding will also depend on social factors. Lower ranking cows may spend less time feeding when they become lame, compared to higher ranking cows, because they will be less able to inhibit the behaviour of other cows and so their choice of foot placement will be less. Unfortunately, no comprehensive studies of the effects of lameness on behaviour have been undertaken. The losses in condition and milk yield may also possibly be due to the effects of lameness itself on the cow's physiology, for example laminitis may lead to circulatory changes which inhibit the supply of metabolites required for milk production from reaching the milk producing tissues. However, this possibility has not been examined.

HOOF TRIMMING

Relative overgrowth of the hind outer claw has been suggested as a predisposing factor in the high incidence of

lameness in this claw (Toussaint-Raven, 1971; 1972; Zantinga, 1972; Noeck, 1972; Andersson, 1980). There is a difference between the outer and inner claw of the hind foot, which is perceptible from the age of 18 to 24 months; the outer claw is slightly larger with a flatter sole and higher heel, and therefore suffers from the effects of excessive weight bearing. Such action of the body weight on the solar corium is thought to be of major importance in the pathogenesis of lameness, and can be alleviated by functional trimming (Toussaint-Raven, 1972).

The main objectives of trimming are the establishment of equal weight bearing between the claws, correction of overburdening of the outer claw, the restoration of a normal bearing surface within each claw and the correction of any developing hoof defects (Toussaint Raven, 1971; 1972; Utrecht University, 1982).

Although trimming has been advocated as an important prophylactic measure in the prevention of solar contusions and solar ulcers through its correction of weight bearing and the prevention of overgrowths (Smedegaard, 1964a; Toussaint-Raven, 1971;1972), Nilsson (1966) suggested that the development of solar ulcers was more dependant on the relationship between laminitis and solar ulcers, in particular the thrombo-embolic formation in the digital vessels in the corium. However, it is more likely that solar ulcers result from a combination of both vessel damage and excessive weight bearing.

Hoof trimming has also been recommended for the prevention of acute laminitis, hoof problems arising both from chronic laminitis and foot rot (Sandelien, 1960; Weaver, 1979), foot rot itself (Smedegaard, 1963) and white line disease (Edwards, 1980). However, Russell et al (1982) found that white line disease occurred equally with normal and overgrown hooves, which indicated that trimming would not have been of use in preventing this disease. Trimming is also thought to hinder curling of the lateral wall under the sole, undue stressing of the weakest part of the sole (the junction of the posterior and middle thirds

of the sole near the axial surface) and cracking at the posterior surface of the heel (Amstutz, 1965).

Although trimming is increasingly being recommended as an effective measure in reducing lameness problems, few trials have been performed to investigate its potential or its relative importance as compared to other prophylactic measures. In a trial involving two herds each containing 120 and 140 spring calving cows, of which half had their hooves trimmed, it was found that trimming did not significantly reduce the incidence of digital diseases (Arkins, 1981). However, the cows feet were trimmed within six weeks of turn out to pasture and since there is an association between the development of solar ulcers and parturition, a beneficial effect may have arisen if trimming was performed in relation to calving date rather than to turn out date. This trial indicates the need for an examination of the optimal time of trimming, if once yearly trimming is carried out. Noordhuizen and Brand (1983) have suggested that trimming should be carried out before cows are turned out to pasture or before a peak in calving pattern, and prior to housing. However, trimming should be avoided during a three month period before cows are put into a new concrete building (Utrecht University, 1982).

The necessity for trimming will vary from herd to herd depending on the feeing regime and housing system in each herd. Therefore, before an assessment of the benefits of trimming on a particular management system can be properly ascertained, an examination of the effects of housing, nutrition and exercise on hoof growth and wear needs to be evaluated.

Dutch trimming is the most widely adopted method and is described in Appendix 2. In general only hind feet need to be trimmed, as there is greater wear in the fore feet, since the toes of the front foot strike the ground first, whereas the heel of the hind foot strikes first (Noeck, 1979).

Thus, there are still large areas of knowledge relevant to a better understanding of lameness which are missing, for example, the relationships between nutrition and lameness. Therefore, experiments were designed to investigate the effects of concentrate level, protein level and concentrate to silage ratio on lameness. The effects of these nutritional variables on various hoof measurements, and the relationships between these measurements and lameness were examined. The effect of Dutch hoof trimming on lameness and on hoof measurements was also investigated. The relationships between behaviour and lameness were also examined so that any effects of lameness on production could be more fully understood.

CHAPTER 2

EXPERIMENT 1. The effect of concentrate level on lameness and hoof shape

INTRODUCTION

Higher levels of concentrate are thought to be associated with a higher incidence of lameness, but few trials have been carried out to verify this. In one trial, a high level of concentrate was found to cause a significantly higher incidence of solar haemorrhage as compared to a lower level; such haemorrhaging was considered to be a preliminary stage of solar ulceration and also a symptom of sub- clinical laminitis (Peterse, 1979). However, the amounts compared, 1 and 6 kg concentrate/day, were not representative of the levels usually fed to high yielding dairy cows.

Clinical incidence has been the main criterion for measuring lameness, but it does not give a true representation of the state of hoof or leg health within a herd or on an individual basis. Therefore, a scoring system was developed whereby every cow could be routinely monitored both for her walking ability and rate of clincial incidence. This system enabled both severity and duration of a hoof or leg problem to be recorded.

Hoof shape is thought to be an important determinant of lameness. Toussaint Raven (1972, 1973) attributed unequal and excessive weight bearing, associated with lameness, to height of claw and differences in claw shape between the outer and inner claw. Toe angle has also been related to wall abnormalities, and hoof length has been associated with the efficiency of the wall to perform its function of transmitting pressure to the skeletal structure (Hahn, 1979). Hoof shape could thus be regarded as a measure of hoof quality.

Therefore, this experiment examined the effects of two concentrate levels, compatible with amounts normally fed

on locomotion and incidence of clinical lameness. The relationships between various hoof shape parameters and locomotion, and the effect of concentrate level on hoof shape were also investigated.

MATERIALS AND METHODS

Treatments

The cattle were balanced for calving date, parity and for the 14 day values of milk yield, liveweight and condition score, and were then allocated at random to the two treatment groups. The 14 day values are shown in Appendix 3. The cattle started on the experiment on average 20 days after calving (range 12 to 27 days).

The two treatments were: 7kg concentrate/day (low) and 11kg concentrate/day (high).

Both concentrate levels were fed at a flat rate (same daily amount to all cows throughout) and silage was offered ad libitum. On average over the trial, the total diet of the low treatment contained 16.8% crude protein and 10.9 MJ/kg DM, and the diet of the high treatment contained 17.5% crude protein and 11.3 MJ/kg DM. The average ratios of silage to concentrate for the low and high groups were 56:44 and 42:58 respectively.

Livestock and management

Forty-eight British Friesian autumn-calving cows, of which twelve were heifers, were used in the continuous design experiment. Their calving dates ranged from 26 August to 17 September. The experiment lasted from weeks 3 to 22 of lactation, and ran from 15 September 1983 to 20 March 1984.

After calving, during the pre-trial period the cattle were housed in a cubicle building, and offered grass silage ad libitum in a feed passage and 6kg concentrates/day in the parlour.

During the trial period the cattle were housed in a cubicle building, the cubicles being bedded with sawdust weekly. The concrete passages were scraped twice daily. The cows walked through a 5% formalin solution footbath weekly.

All cattle received 1kg of the concentrate at the am and pm

milking. The remaining concentrate (5 and 9kg/day for the low and high groups, respectively) was fed from out-of-parlour feeders sited in the housing area, which were programmed to dispense the concentrate allowance equally in four, six hour periods. All the heifers were initially trained in the use of the feeders. Those cows showing refusals on the control box were also trained. Both in and out-of-parlour feeders were calibrated weekly, and any concentrate refusals from the out-of-parlour feeders were recorded daily. The concentrate fed in the parlour and out-of-parlour feeders was in a pelleted form.

The silage was made from first cut perennial ryegrass, cut on the 28 May 1983 with a drum mower, wilted for 24 hours and harvested with a precision chop forage harvester. Formic acid (Add-F, BP Interational Ltd., 870g formic acid/l) was applied at 3.0 litres/tonne to the silage, which was ensiled in an unroofed silage bunker and sheeted with black polythene. The silage was group fed to each treatment, and refusals of silage were recorded twice weekly. The method used to calculate individual silage intakes from weekly group silage intakes is described in Appendix 4.

The chemical compositions of the silages and concentrate are shown in Table 2.10. The physical ingredients of the concentrate are shown in Table 2.11.

Methods and records

Silage was sampled for dry matter daily and concentrates weekly. Weekly samples of the silage and concentrate were also taken for chemical analysis. The techniques and equations for these analyses were those of Alexander and McGowan (1966; 1969) (see Appendix 5).

Milk yields were recorded weekly and milk samples taken for the determinations of fat, protein and lactose contents, from which solids-not-fat and total solids contents were calculated. A Foss Electric Milkoscan (Briggs,1979) was used for analyzing the

Table 2.10 Chemical Composition of Feeds (g/kg DM)

	Silage	Concentrate
Oven dry matter (g/kg)	190	864
Crude protein	146	196
Organic matter	887	898
D value	611	704
Predicted ME (MJ/kg DM)	9.8	12.4
Ammonia N (g/kg total N)	125	
pH	4.1	
Calcium	5.2	13.9
Phosphor us	3.5	8.0
Magnesium	2.0	7.5

Table 2.11 Physical Ingredients of Concentrate (kg/1000 kg)

Barley	243
Maize gluten	202
Wheat	200
Wheat feed	80
Soya	150
Fishmeal	25
Molasses	50
Fat supplement	20
Dicalcium phosphate	5
Minerals and vitamins	25

samples. Liveweight and condition scores were recorded weekly after the pm milking. The tail head system of Mulvany (1977) was used for condition scoring. In addition to lameness, records of mastitis and other aspects of health were kept.

Locomotion scores were recorded weekly before the pm milking. The cows were observed from behind while they walked a distance of about 10 metres on the concrete loafing area, and were scored on a scale of 1 to 5 with half scores. Scores of 3 and above indicated lameness, and all cows scoring 3 or more were examined by a trained member of staff, diagnosed and treated as necessary. Each time a cow scored 3 or more she was counted as being clinically lame even if she had been treated previously. Although in some cases a repeated solar problem or a repeated solar problem in another claw may have been a manifestation of the same common primary (laminitic) process, more cases of clinical lameness per cow could be regarded as an indication of the severity of the the diseases. Details of the locomotion scoring are given in Appendix 6.

The models on which the estimates of repeatability were based were those of Hahn (1979). To estimate the repeatability of locomotion scores made on the same cow by the same observer, a group of 96 cows was scored for locomotion on 2 different occasions separated by a 90 minute interval. The repeatability was calculated using the following equation:

$$r=var(cow)/\{var(cow)+var(record)+var(error)\},$$

where cow is one of the cows measured and record is the effect of the first or second measurement. To estimate the repeatability of locomotion scores made on the same cows by different observers, a group of 96 cows was scored for locomotion by 3 different observers on 2 different occasions separated by a 90 minute interval. Each observer was initially trained in locomotion scoring during a two hour period. Subsequently each observer was cross checked with a trained observer monthly over a four month period. The repeatability was calculated using the following equation:

$$r=var(cow)/\{var(observer)+var(record)+var(observerxrecord)+ var(cow)+var(observerxcow)+var(error)\},$$

where observer is the effect of the 3 individual observers.

Hoof shape measurements were recorded at 3 weeks post calving, and further measurements were recorded 10 and 20 weeks later. The measurements, which were taken in the parlour, were recorded on the right hind foot and were as follows:

length of toe of outer and inner claw (lot, lit)

angle of toe of outer and inner claw (aot, ait)

height of heel of outer claw (hoh).

The height of heel of the inner claw was not measured, since it was difficult to determine the position of the basis of the bulb, visual judgement being impossible. For measurement positions, see Figure 2.1. Prior to measuring, the hooves were brushed and washed to remove any dirt.

The nomenclature used for describing the hoof is that used by the European Association of Animal Production (Empel et al, 1983). The length of toe (distance from the top of the dorsal wall to the distal edge of the claw) was measured using a standard divider and ruler. The angle of toe (angle of the dorsal side of the wall with respect to the ground) was measured using a commercial protractor (Universal Protractor, Sears, Roebuck and Co., Chicago, Il.), modified by glueing a small wooden block to the base. The protractor was placed equi-distant between the top of the dorsal wall and the distal edge of the claws. The height of heel (perpendicular distance from the basis of the bulb bulb to the ground) was measured using a sliding ruler.

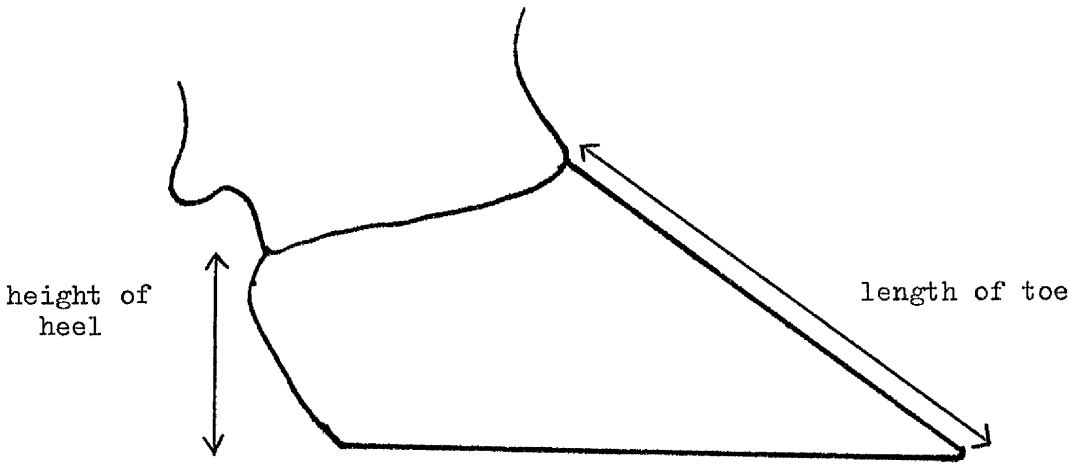
To estimate the repeatability of hoof shape measurements made on the same cow by the same observer, the group of 48 cows used in the experiment were recorded for each hoof shape measurement on 2 occasions, separated by an interval of 60 minutes. The repeatability was calculated using the following equation:

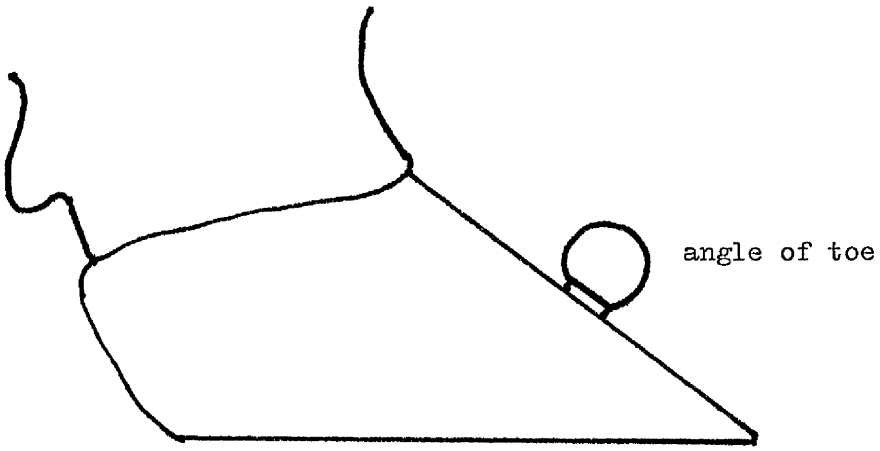
$$r = var(cow)\{var(cow)+var(record)+var(error)\}.$$

Statistical analysis

The results were analysed as a randomised block design using the statistical package Genstat 5 Mark 4.03 (Lawes Agricultural Trust 1980). Minitab (Pennyslyvania State University, 1980) was

Figure 2.1 Hoof measurements





used to calculate correlation and regression coefficients between various measurements.

Data for cows and heifers were analysed in successive four, five week periods. Chi- squared analysis was used to investigate the assocation between treatments and clinical incidence of lameness to determine whether it was necessary to adjust a current incident of lameness for a particular cow, for a previous incident of lameness for that same cow. An analysis of variance was carried out. In this analysis the following sources of variation were used: number of weeks on trial, number of treatments, number of cows in each treatment, number of treatment weeks, experimental error and a covariate. The analysis was adjusted for the covariate, which was for whether a particular cow had been previously lame or not. "Previous" meant within the previous 8 weeks, for example, if a cow who was currently lame had been lame say 10 weeks previously, then her current incident of lameness would not have been adjusted for her previous lameness. A period of 8 weeks was chosen, as this was considered to be sufficient time for a cow to recover from a solar ulcer, foot rot etc.. It was found that the adjustment using the covariate made no significant improvement to the analysis, and it was concluded that current lameness could be regarded as being independant of previous lameness.

RESULTS

Data are presented as four, five week periods, commencing at three weeks post calving.

Locomotion scores and clinical incidence of lameness

Although the frequency distribution curve for locomotion scores was slightly skewed, the locomotion score results were analysed as for a normal distribution. No suitable transformation whereby the variance was constant for all the experimental units was found, and it was concluded that the most appropriate method was to treat the locomotion scores as being normally distributed.

Locomotion scores were significantly higher for the high concentrate group in the first ($p<0.05$) and fourth periods

($p<0.01$), but the mean locomotion scores over the whole experiment were not significantly different ($p>0.05$) (see Table 2.12). There were no particular trends with stage of lactation, although there was a tendency for lower scores to be obtained in the later part of the trial.

The repeatability of a cows locomotion score assessed on 2 occasions by the same observer (within observer repeatability) was 0.89, and the repeatability of a cow's score assessed by the 3 observers on 2 occasions (across observer repeatability) was 0.84. Concentrate level was significantly ($p<0.001$) associated with the number of clinical cases per cow week, with a higher incidence of lameness in the high concentrate group (see Table 2.13). Fifteen per cent of lameness cases were due to leg lesions and 85% due to foot lesions, of which 95% occurred in the hind hooves (see Table 2.14). The majority of lameness cases were due to solar problems (see Table 2.15).

Hoof measurements

The initial outer toe lengths and initial outer heel heights were significantly ($p<0.05$) longer in the high group. The differences in initial toe angle were not significant. Differences for all subsequent hoof measurements were also not significant (see Table 2.16).

There was significantly more change (net growth) between the inital and 10 week measurements for the outer ($p<0.01$) and inner ($p<0.05$) toe lengths in the low group. There were non significant differences for the changes (increases or decreases) in toe angle and heel height over this period, and for all the changes in hoof shape between weeks 10 to 20, and between the initial and 20 week measurements (see Table 2.17). Changes on the hoof shape measurements were calculated from unadjusted figures.

There was a negative correlation between toe length and toe angle for both outer and inner claws. However, toe length could not be used as a predictor of toe angle, since significant correlations were found only between the measurements taken at 20 weeks for the outer ($p<0.001$)and inner ($p<0.01$) claws, and for

Table 2.12 Locomotion score means

	Treatments		
	Low	High	SED
Period 1			
Locomotion score	1.57	1.79	0.10 *
Period 2			
Locomotion score	1.43	1.66	0.12 NS
Period 3			
Locomotion score	1.45	1.65	0.12 NS
Period 4			
Locomotion score	1.34	1.62	0.09 **
Mean of 1 to 4			
Locomotion score	1.45	1.68	0.09 *

Table 2.13 Incidence of lameness

	Treatments	
	Low	High
Number of clinical cases per cow week*	0.021	0.077

concentrate level *** (Chi-squared analysis)

* cows scoring 3 or more divided by the number of cow weeks (12x24)

Table 2.14 Number of clinical cases @ (weekly recordings) in relation to the site of the lesion

	Treatments				
	Low		High		
	leg lesion	claw lesion	leg lesion	claw lesion	totals
Right hind	2	5	2	16	25
Left hind	0	1	1	16	18
Right fore	0	2	1	0	3
Left fore	0	0	1	0	1
totals	2	8	5	32	47

@ cows with locomotion scores of 3 or more

Table 2.15 Diagnoses of clinical cases of lameness $

	Treatments	
	Low	High
Number of examinations	10	37
Solar problem	7	28
Interdigital growth	0	4
Foot rot@	1	0
Leg injury	2	5

$ cows with locomotion scores of 3 or more

@ foot rot=Dermatitis interdigitalis with underrunning

Table 2.16 Hoof shape means @

	Treatments		
	Low	High	SED
Initial measurements			
Length of toe, outer (cm)	7.4	7.9	0.22 *
Length of toe, inner (cm)	7.4	7.9	0.21 *
Angle of toe, outer (o)	37	37	2.22 NS
Angle of toe, inner (o)	44	45	1.22 NS
Height of heel, outer, (cm)	3.1	3.4	0.14 *
Measurements at 10 weeks			
Length of toe, outer (cm)	7.9	7.7	0.19 NS
Length of toe, inner (cm)	7.7	7.5	0.21 NS
Angle of toe, outer (o)	38	38	2.56 NS
Angle of toe, inner (o)	48	48	1.50 NS
Height of heel, outer (cm)	4.0	3.9	0.18 NS

@ means adjusted for covariates, which are the initial measurements

Table 2.16 Hoof shape means @

(continued)

	Treatments		
	Low	High	SED
Measurements at 20 weeks			
Length of toe, outer (cm)	8.1	8.1	0.23 NS
Length of toe, inner (cm)	7.7	8.0	0.22 NS
Angle of toe, outer (o)	38	37	2.39 NS
Angle of toe, inner (o)	48	47	1.18 NS
Height of heel, outer (cm)	4.0	3.9	0.13 NS

@ means adjusted for the covariates, which are the initial measurements

Table 2.17 Changes in hoof shape means @

	Treatments		
	Low	High	SED
Changes from initial to 10 week measurements			
Length of toe, outer (cm)	+0.54	-0.22	0.25 **
Length of toe, inner (cm)	+0.18	-0.31	0.22 *
Angle of toe, outer (o)	+1.5	+0.7	2.51 NS
Angle of toe, inner (o)	+3.4	+3.3	1.53 NS
Height of heel, outer (cm)	+0.93	+0.57	0.19 NS
Changes from 10 week to 20 week measurements			
Length of toe, outer (cm)	+0.15	+0.39	0.17 NS
Length of toe, inner (cm)	+0.13	+0.34	0.21 NS
Angle of toe, outer (o)	-0.8	-0.9	1.67 NS
Angle of toe, inner (o)	-0.1	-1.2	1.45 NS
Height of heel, outer (cm)	-0.03	-0.06	0.15 NS

@ changes calculated from unadjusted hoof shape means

Table 2.17 Changes in hoof shape means@

(continued)

	Treatments		
	Low	High	SED
Changes from initial to 20 week measurements			
Length of toe, outer (cm)	+0.69	+0.17	0.29 NS
Length of toe, inner (cm)	+0.31	+0.03	0.29 NS
Angle of toe, outer (o)	+0.7	-0.2	2.37 NS
Angle of toe, inner (o)	+3.3	+2.1	1.19 NS
Height of heel, outer (cm)	+0.88	+0.53	0.18 NS

@ changes calculated from unadjusted hoof shape means

Table 2.18 Correlations and regression equations for hoof shape measurements

Mean angle of toe, outer (y) on mean length of toe, outer (x)

initial	week 10	week 20
r=-0.236 NS	r=-0.098 NS	r=-0.581 ($p<0.001$)
$y=49.9-1.7(\pm 1.04)x$	$y=47.2-1.2(\pm 1.76)x$	$y=105.3-8.4(\pm 1.74)x$

Mean angle of toe, inner (y) on mean length of toe, inner (x)

initial	week 10	week 20
r=-0.197 NS	r=-0.299 ($p<0.05$)	r=-0.465 ($p<0.01$)
$y=52.7-1.1(\pm 0.08)x$	$y=63.7-2.1(\pm 0.99)x$	$y=72.5-3.3(\pm 0.91)x$

Mean height of heel, outer (y) on mean length of toe, outer(x)

initial	week 10	week 20
r=+0.111 NS	r=+0.182 NS	r=-0.032 NS
$y=2.74+0.06(\pm 0.08)x$	$y=2.87+0.14(\pm 0.11)x$	$y=4.10-0.02(\pm 0.10)x$

Table 2.19 Differences between outer and inner claw means

	Treatments		
	Low	High	SED
Initial measurements			
Difference between outer and inner claw toe length (cm)	0.49	0.49	0.10 NS
Difference between outer and inner claw toe angle (o)	8.1	7.7	1.53 NS
Measurements at 10 weeks			
Difference between outer and inner claw toe length (cm)	0.43	0.51	0.11 NS
Difference between outer and inner claw toe angle (o)	9.3	10.3	2.23 NS
Measurements at 20 weeks			
Difference between outer and inner claw toe length (cm)	0.49	0.43	0.08 NS
Difference between outer and inner claw toe angle	10.4	9.4	2.39 NS

measurements taken at 10 weeks for the inner claw ($p<0.05$). No significant correlations between toe length and heel height were found (see Table 2.18).

The magnitude of the differences in toe length and toe angle between the outer and inner claws, was not significantly different between the high and low concentrate groups (see Table 2.19).

The within observer repeatabilities of the hoof shape measurements were as follows: length of outer toe, 0.92; length of inner toe, 0.90; angle of outer toe, 0.89; angle of inner toe, 0.86 and height of outer heel, 0.75.

Feed intake

The mean intakes of concentrate DM, silage DM, total DM and estimated ME and CP intakes are shown in Table 2.20. The silage intakes of the high group were significantly lower than those of the low group in each period. The mean substitution rate was 0.43kg reduction in silage DM intake per kg increase in concentrate dry matter intake.

Cow performance

In all four periods the milk yields of the high group were significantly ($p<0.001$) higher than those of the low group (see Table 2.21). The mean fat and lactose contents of the milk were significantly ($p<0.01$) less and significantly ($p<0.001$) more, respectively, in the high group. The mean protein contents of the milk were not significantly affected by the level of concentrate input. Mean solids-not fat contents were significantly ($p<0.01$) higher in the high group. The differences in mean liveweight and mean liveweight change between the two groups were not significantly different. Condition scores were significantly ($p<0.05$) higher on the high concentrate groups (see Tables 2.22 to 2.24).

Table 2.20 Mean daily intakes of dry matter and the calculated Metabolisable Energy and Crude Protein intakes

	Treatments		
	Low	High	SED
Period 1			
Silage DM intake (kg/day)	8.7	6.6	0.33 ***
Concentrate DM intake (kg/day)	6.1	9.3	0.13 ***
Total DM intake (kg/day)	14.8	15.9	
Total ME intake (MJ/day)	161	180	3.56 ***
Total CP intake (g/day)	2459	2793	53.96 ***
Conc:forage ratio	41:59	58:42	
Period 2			
Silage DM intake (kg/day)	8.5	7.2	0.38**
Concentrate DM intake (kg/day)	6.2	9.4	0.13***
Total DM intake (kg/day)	14.7	16.6	
Total ME intake (MJ/day)	160	188	3.98 ***
Total CP intake (g/day)	2445	2909	59.40 ***
Conc:forage ratio	42:58	56:44	

Table 2.20 Mean daily intakes of dry matter and the calculated (continued) Metabolisable Energy and Crude Protein intakes

	Treatments		
	Low	High	SED
Period 3			
Silage DM intake (kg/day)	7.2	6.0	0.41 **
Concentrate DM intake (kg/day)	6.2	9.3	0.07 ***
Total DM intake (kg/day)	13.4	15.3	
Total ME intake (MJ/day)	147	174	4.25 ***
Total CP intake (g/day)	2261	2697	62.87 ***
Conc:forage ratio	46:54	61:39	
Period 4			
Silage DM intake (kg/day)	7.7	6.8	0.41 *
Concentrate DM intake (kg/day)	6.2	9.3	0.07 ***
Total DM intake (kg/day)	13.9	16.1	
Total ME intake (ME/day)	153	182	4.27 ***
Total CP intake (g/day)	2351	2813	63.81 ***
Conc:forage ratio	45:55	58:42	

Table 2.20 Mean daily intakes of dry matter and the calculated (continued) Metabolisable Energy and Crude Protein intakes

	Treatments			
	Low	High	SED	
Mean of 1 to 4				
Silage DM intake (kg/day)	8.0	6.7	0.29	***
Concentrate DM intake (kg/day)	6.2	9.3	0.06	***
Total DM intake (kg/day)	14.2	16.0		
Total ME intake (MJ/day)	155	181	3.12	***
Total CP intake (g/day)	2379	2803	47.20	***
Conc:forage ratio	44:56	58:42		

Table 2.21 Milk yield means

	Treatments		
	Low	High	SED
Period 1			
Milk yield (kg/day)	24.4	26.5	0.55 ***
Period 2			
Milk yield (kg/day)	21.5	25.2	0.53 ***
Period 3			
Milk yield (kg/day)	18.5	22.5	0.51 ***
Period 4			
Milk yield (kg/day)	17.5	20.8	0.80 ***
Mean of 1 to 4			
Milk yield (kg/day)	20.5	23.7	0.51 ***

Table 2.22 Milk composition means (g/kg)

	Treatments		
	Low	High	SED
Period 1			
Milk fat content	40.0	37.5	0.90 *
Milk protein content	30.9	31.0	0.52 NS
Period 2			
Milk fat content	42.3	39.5	1.22 *
Milk protein content	31.1	31.6	0.62 NS
Period 3			
Milk fat content	42.5	39.5	1.00 **
Milk protein content	31.3	32.4	0.62 NS
Period 4			
Milk fat content	42.5	39.8	1.12 *
Milk protein content	32.6	33.2	0.80 NS
Mean of 1 to 4			
Milk fat content	41.7	39.0	0.92 **
Milk protein content	31.4	32.0	0.60 NS
Milk lactose content	48.6	49.7	0.23 ***
Milk s-n-f content	87.3	89.4	0.61 **
Milk total solids content	128.9	128.4	1.33 NS

Table 2.23 Liveweight and liveweight change means

	Treatments			
	Low	High	SED	
Period 1				
Liveweight (kg)	563	567	5.65	NS
Liveweight change (kg/day)	+0.18	-0.20	0.17	*
Period 2				
Liveweight (kg)	573	577	6.40	NS
Liveweight change (kg/day)	-0.06	-0.32	0.14	NS
Period 3				
Liveweight (kg)	565	569	6.38	NS
Liveweight change (kg/day)	-0.14	-0.36	0.17	NS
Period 4				
Liveweight (kg)	569	574	8.20	NS
Liveweight change (kg/day)	+0.45	+0.28	0.12	NS
Mean of periods 1 to 4				
Liveweight (kg)	568	572	6.21	NS
Liveweight change (kg/day)	+0.11	-0.12	0.11	NS

Table 2.24 Condition score means

	Treatments		
	Low	High	SED
Period 1			
Condition score	2.09	2.11	0.06 NS
Period 2			
Condition score	2.01	2.19	0.07 *
Period 3			
Condition score	2.05	2.24	0.08 *
Period 4			
Condition score	2.02	2.19	0.07 *
Mean of 1 to 4			
Condition score	2.04	2.18	0.06 *

DISCUSSION

The lack of a major lameness problem in either group as indicated by the low locomotion scores, was possibly due to the distribution of the concentrate feed into 6 portions a day, and ready access to ad libitum silage. Higher feeding frequencies of concentrates are thought to reduce the incidence of acidosis, which would in turn decrease the likelihood of laminitis occurring (Kaufmann, 1976; Weaver, 1979). Trimming of the cows feet prior to housing, regular foot-bathing and adequate housing would also have contributed to the low incidence of lameness. However, the scores obtained for the high group in the first period may be evidence of the presence of sub-clinical laminitis in some of the cows. Mean scores of about 1.75 are reflections of the symptoms of sub-clinical laminitis, for example, abduction, adduction and uneveness (Appendix 6). Additionally many of the clinical cases were due to solar problems, which are often the sequelae of laminitis (Nilsson, 1963; 1966; Toussaint-Raven, 1972, 1973).

Whether the effect of feeding a high concentrate level in causing a significantly higher locomotion score in the first and fourth periods was attributable to the higher CP intake, higher ME intake or higher concentrate to forage ratio is unknown, since these three parameters were confounded in the trial. However, the significant difference in locomotion score between the groups in the first period, coincides with the largest difference in concentrate to forage ratio between the groups. The concentrate to forage ratio is probably important through its effects on acidosis and laminitis. The increase of concentrate level in relation to forage level, with its subsequent decreases in ruminal pH, as an etiological factor in acidosis and laminitis has been well documented (Nilsson, 1963; Brent, 1976; Kaufmann, 1976; Weaver, 1979; Livesey and Fleming, 1984).

Laminitis has also been observed to appear most frequently at parturition or during the following month (Nilsson, 1963, 1966; Weaver, 1979; Greenough et al, 1981), which corresponds to Period 1. Locomotion scores were highest for both groups in this period

and then decreased, which probably indicated that there was some recovery from laminitis rather than a progression into a chronic phase. Maclean (1965) speculated that parturition may supply the necessary stimuli to allow the absorption of histamine, which has been implicated in the onset of laminitis, from the alimentary tract into the blood stream. The tendency for cows to contract laminitis around parturition may have been exacerbated by the higher concentrate to forage ratio in the high concentrate group.

The significantly higher locomotion scores of the high group in the final period may have been caused by the slower recovery rate from hoof problems, which were most prevalent in the first period. These smaller decreases in locomotion score over the trial period, as compared to the low group, were probably due to a more severe and a higher incidence of laminitis around calving.

The significant difference in the final period may also partly be explained by the differences in relationship between hoof shape and locomotion score between the groups at 20 weeks. In the high group inner claw toe length and angle were significantly ($p<0.05$) correlated with locomotion score, see equations 2.1 and 2.2:

$r=+0.51$ ($p<0.05$)----------equation 2.1

$y=-2.62+0.56(\pm 0.20)x$

where y=mean locomotion score and

x=inner claw toe length

$r=-0.55$ ($p<0.01$)----------equation 2.2

$y=5.62-0.08(\pm 0.03)x$

where y=mean locomotion score and

x=inner claw toe angle

In the low group correlations and regressions between hoof shape and locomotion score were not significant at 20 weeks. Therefore, since the differences between the low and high groups in toe length and angle at 20 weeks were not significant, the cows in the high group would have contributed proportionately more to their locomotion score than those with similarly lengthed toes in the low group. For example, a cow in the high group with an inner claw toe length of 10 cm, as compared to a cow with a toe length

of 7 cm would have an increase in locomotion score of 1.68 units, whereas in the low group the equivalent increase would only be 0.18 locomotion units. The higher levels of variation both in locomotion score and in toe length in the high concentrate group, as compared to the low concentrate group, may have accounted for there being a correlation between locomotion score and toe length in the high concentrate group but not in the low concentrate group.

Although the toe lengths were initially significantly less in the low group, the differences between the groups at weeks 10 and 20 were not significant. This can be ascribed to the significantly greater change (net growth) for the outer ($p<0.01$) and inner claw ($p<0.05$) toe lengths in the low group from the initial to the 10 week measurements. In the high group over this period there was net wear. The non significant differences in toe length change from weeks 10 to 20, would have accounted for the continuance of an absence in toe length difference between the groups at 20 weeks.

A higher concentrate level, by increasing the concentrate to forage ratio, may decrease net growth through it predisposing to laminitis. Pathological changes occurring during laminitis include congestion of the corium, loss of capillary integrity and bypassing of the capillaries supplying the corium with subsequent blood stagnation. These changes would lead to an insufficient nutrient supply to the horn producing cells and the formation of structurally incompetent horn (Coffman, 1970; Greenough et al, 1981; Edwards, 1982). Since the horn produced during laminitis would be of poorer quality and probably softer and less elastic, the horn reaching the distal surface would be more liable to wear Consequently a reduction in net hoof growth might be expected. Therefore, the significantly higher level of net growth from the initial to the 10 week measurements in the low group, could be explained by the lower levels of laminitis, which is also reflected in the lower locomotion scores of the low group. Elongated toes have been associated with chronic laminitis (Prentice, 1970; Livesey and Fleming, 1984). However, the levels of laminitis in this trial were low and were probably sub-acute in nature. This together with the likelihood of a relatively fast recovery from

laminitis, as indicated by the decline in locomotion scores in the high concentrate group over the trial period, may have prevented such overgrowths from occurring. However, further investigation into the effects of concentrate to forage ratio on growth and hoof wear, separately, is required before any firm conclusions can be drawn.

The non significant differences between groups in changes of heel height may be explained by the apparent absence of an effect of high concentrate levels in predisposing to Dermatitis interdigitalis which is characterised by an erosion of the heel.

The finding that the differences between the outer and inner claws in toe length and angle, were not significantly different between the groups, indicates that overloading of the corium, which is partly caused by unequal weight distribution within the claws, was probably of a similar magnitude in both groups. The toe lengths, toe angles and heel heights on both groups were also of a magnitude unlikely to cause weight distribution problems within the claws. These findings in conjunction with the non significant differences between hoof shape at 10 and 20 weeks, may suggest that the differences in locomotion score between the groups were mainly due to nutritionally related problems, such as laminitis, rather than to hoof shape factors.

The high response in milk yield of 1.02kg for each kg increase in concentrate DM, was probably due to the small substitution effect of concentrate DM for a silage DM that had a low predicted ME (9.8 MJ/kg). The significantly lower fat contents and significantly higher lactose contents in the high concentrate group are consistent with the findings of Moisey and Leaver (1985). The lack of a significant effect of concentrate level on mean protein content agrees with the results of Gordon (1977).

The correlations between milk yield and locomotion score were not significant. This was probably due to the small variation in locomotion scores, and to the low level of incidence of clinical lameness having a minimal effect on food intake and therefore on subsequent milk production.

Feeding concentrate at a high level probably increased the

incidence of lameness and locomotion scores through the high concentrate to forage ratio predisposing to laminitis around calving. Confirmation of this is needed, since the high levels of ME and CP may also have been important. However, the high concentrate level did not lead to a serious lameness problem, probably because the concentrate to forage ratio was not extreme and the concentrate was fed at a high frequency. Although concentrate level did not significantly affect hoof shape, there was some evidence of an effect on growth and wear and this aspect requires further investigation.

It may be concluded from the within observer repeatabilities of locomotion score that locomotion scoring could be used with reasonable precision. The across observer repeatabilities indicated that differences due to observers were not particularly important as a source of variation in comparison to that attributable to between cow differences. The finding that the within observer repeatabilities were higher than the across observer repeatabilities suggested that whilst observers may be consistent in their scoring, each observer may score consistently very slightly higher or lower as compared with another observer.

The repeatabilities of the hoof shape measurements compared favourably with those of Hahn (1979) and Huber (1983), who found repeatabilities between 0.76 and 0.95 for toe length, between 0.76 and 0.90 for toe angle and between 0.68 and 0.80 for height of heel. The repeatabilities were probably influenced by ease of locating the reference marks used for each measurement, and the cows standing position. For example, the proximal mark of the heel was more difficult to find compared to that of the toe.

SUMMARY

Two groups of 18 cows and 6 heifers were offered two levels of concentrate (7 versus 11 kg/day), fed at a flat rate, during weeks 3 to 22 of lactation. Silage was available ad libitum. The cows were scored for their locomotion weekly on a 1 to 5 scale (high score indicated poorer locomotion). Hoof shape measurements were recorded at the beginning, mid point and end of the trial. Outer and inner claw toe lengths and angles, and outer claw heel heights were measured on the right hind hoof.

The cows on the high concentrate level had significantly greater locomotion scores, compared to those on the low concentrate level. There was also a higher incidence of clinical lameness in the high concentrate group, the major problem being solar ulceration.

The hoof shape measurements for the two groups were not significantly different.

The correlations within treatments between levels of milk production and locomotion score were negative although non significant.

CHAPTER 3

EXPERIMENT 2. The effect of protein level and Dutch hoof trimming on lameness, hoof growth and wear, hoof shape and hoof hardness

In Experiment 1 the higher concentrate level was associated with higher locomotion scores and a greater incidence of clinical lameness. These may have resulted from the higher protein or energy intakes or from the higher concentrate to silage ratio. The factors which are of importance in the development of lameness need to be identified.

Some authors have suggested that a protein related histaminotic phenomenon may be involved in the etiology of laminitis (Akerblom, 1934; Nilsson, 1963; Maclean, 1966; Morrow, 1966), whilst others (Urmas, 1968; Chew, 1972) have implicated toxins of proteinaceous origin of being of importance. These suggestions were based on cows who were already diagnosed as being lame. However, to identify whether protein is important in the development of lameness, it would also be necessary to compare the effects of different protein levels and sources on groups of cows who were not initially lame.

There were indications that locomotion score was related to toe length and angle in the high concentrate group in Experiment 1, and therefore trimming and relative rates of hoof growth and wear are likely to be important factors in the predisposition of a cow to lameness.

Hoof hardness measures the resistance of horn to penetration, and is therefore likely to be related to hoof problems such as septic penetrations and solar ulcers. Additionally hardness measurements may indicate the ability of the heel bulb to function correctly. A soft heel bulb would be desirable, since the bulb needs to act as a cushion, spreading the load evenly. As levels of sulphur amino acids are important in determining the hardness of the hoof (Clark and Rakes, 1982), protein levels are likely to be of importance.

The relationships between levels of certain blood parameters

with locomotion score may give some indication of the etiological factors involved in laminitis, and of the potential use of blood analyses as a diagnostic aid for laminitis.

This experiment investigated the effects of protein level and Dutch hoof trimming on locomotion, incidence of clinical lameness, hoof growth, wear, shape and hardness. The relationships between the hoof measurements, various blood parameters and lameness were also examined.

MATERIALS AND METHODS

Treatments

Thirty-six cows were allocated in quartets according to parity and projected calving date, four months before the trial started. Two cows from each quartet were chosen at random and had their feet trimmed to Dutch standards. The Dutch trimming method is described in Appendix 2. Ten days after calving six heifers from a group of twelve were chosen and had their hooves trimmed. Quartets of cows containing two trimmed and two untrimmed animals were balanced for the 14 day values of milk yield and liveweight. The 14 day values are shown in Appendix 7. Each of the two trimmed cows were allocated at random to one of the two dietary treatments and the untrimmed cows were similarly allocated. The cattle started on the experiment on average 17 days after calving (range 11 to 26 days). The trial lasted from 13 September, 1984 to 4 April, 1985.

The four treatments were:

161 g/kg crude protein in the total dry matter, hooves trimmed (LT)

161 g/kg crude protein in the total dry matter, hooves untrimmed (LUT)

198 g/kg crude protein in the total dry matter, hooves trimmed (HT)

198 g/kg crude protein in the total dry matter, hooves untrimmed (HUT).

On average over the whole trial both the low and high protein diets contained 12.2 MJ/kg DM. The concentrate to silage ratio in the total diet was 60:40 on a dry matter basis for both diets.

All cattle received 1 kg concentrate at the am and pm milking. The remainder of the diet was fed as a complete diet and the silage and protein mixes were mixed using a paddle type West mixer wagon. The protein levels were adjusted by substituting

soya for sugar beet. Mean ME intakes were maintained at the same level for all treatments by adjusting twice weekly the high protein diet intake to that of the low protein diet, which was fed ad libitum.

Livestock and management

Forty-eight British Friesan cows, of which twelve were heifers, were used in a continuous design experiment, which lasted from weeks 3 to 26 of lactation. The calving dates ranged from 26 August to 17 September. The experiment ran from 13 September 1984 to 4 April 1985.

After calving during the pre-trial period, the cattle were housed in a cubicle building, and offered grass silage ad libitum in a feed passage and 6 kg concentrates/day in the parlour.

During the trial period the cattle were housed in a cubicle building, the cubicles being bedded with sawdust weekly. The concrete passages were scraped twice daily. The cows walked through a 5% formalin footbath weekly.

Three batches of silage made from perennial ryegrass were used. The initial batch of first cut silage was cut on the 29 May 1984, and fed for the first 7 weeks. The second batch of first cut silage was cut on the 29 May 1984, and fed from weeks 8 to 19, and the third batch of second cut silage was cut on the 4 July 1984, and fed for the remainder of the trial period. All the batches were cut with a drum mower, wilted for 24 hours and harvested with a precision chop forage harvester. Formic acid (Add-F, BP International Ltd., 850 g formic acid/l) was applied at 2.3 litres/tonne to all the batches, which were ensiled in unroofed silage bunkers and sheeted with black polythene.

The chemical compositions of the silages, protein mixes, concentrate, and of the total low and high protein diets are shown in Tables 3.10 to 3.12. The physical ingredients of the concentrate and and protein mixes are shown in Table 3.13.

Table 3.10 Chemical Composition of Silages (g/kg DM)

	First batch	Second batch	Third batch	Mean of batches 1 to 3
Oven dry matter (g/kg)	286	256	290	277
Crude protein	178	157	196	177
Rumen degradable protein@	151	135	166	151
Undegradable protein@	27	24	29	27
Organic matter	905	926	908	913
DOMD (in vitro)	678	721	683	694
Predicted ME (MJ/kg DM)	10.7	11.4	10.8	11.0
Ammonia N (g/kg total N)	102	90	101	98
pH	4.4	4.1	4.2	4.2
Calcium	6.8	5.6	7.4	6.6
Phosphor us	3.5	3.0	3.1	3.2
Magnesium	2.9	2.4	3.3	2.9

@ degradabilities based on theoretical values

Table 3.11 Chemical compositions of protein mixes and concentrate (g/kg DM)

	concentrate	low protein mix	high protein mix
Oven dry matter (g/kg)	861	864	865
Crude protein	195	141	216
Rumen degradable protein@	133	103	158
Undegradable protein@	62	38	58
DOMD (in vitro)	754	804	809
Predicted ME (MJ/kg DM)	13.5	12.8	12.9
Calcium	10.6	9.6	9.8
Phosphor us	7.7	5.9	6.9
Magnesium	5.7	5.4	4.9

@ degradabilities based on theoretical values

Table 3.12 Chemical compositions of total diets (g/kg DM)

	Low protein diet	High protein diet
Oven dry matter (g/kg)	629	629
Crude protein	161	198
Rumen degradable protein@	125	153
Undegradable protein@	36	46
DOMD (in vitro)	755	758
Predicted ME (MJ/kg DM)	12.2	12.2
Calcium	8.5	8.6
Phosphorous	5.0	5.5
Magnesium	4.4	4.2

@ degradabilities based on theoretical values

Table 3.13 Physical Ingredients of Concentrate (kg/1000 kg)

Barley	250
Maize gluten	200
Wheat	200
Wheat feed	80
Soya	150
Fishmeal	20
Molasses	50
Fat supplement	20
Dicalcium phosphate	5
Minerals and vitamins	25

Physical ingredients of protein mixes (kg/1000 kg)

	Low protein mix	High protein mix
Barley	614	614
Soya	39	234
Sugar beet	312	117
Minerals and vitamins	35	35

Methods and records

Cow performance

The protein mixes and the parlour concentrate were sampled for dry matter weekly and the silage daily. Weekly samples of the silage, protein mixes and concentrate were also taken for chemical analysis. The techniques and equations for these analyses were those of Alexander and McGowan (1966; 1969), (see Appendix 5).

Milk yields were recorded weekly and milk samples taken for the determinations of fat, protein and lactose contents, from which solids-not-fat and total solids contents were calculated. A Foss Electric Milkoscan (Briggs,1979) was used for analysing samples. Liveweight and condition scores were recorded after the the pm milking weekly. The tail head system of Mulvany (1977) was used for condition scoring. In addition to lameness, records of fertility, mastitis and other aspects of health were kept.

Locomotion scoring and hoof measurements

Locomotion scores were recorded weekly before the pm milking. The cows were observed from behind while they walked a distance of about 10 metres on the concrete loafing area, and were scored on a scale of 1 to 5 with half scores. Scores of 3 and above indicated lameness, and all cows scoring 3 or more were examined, diagnosed and treated as necessary. Details of the locomotion scoring are given in Appendix 6.

Hoof shape measurements were recorded on the right hind hoof and were as follows:

length of toe of outer and inner claw (lot, lit)
angle of toe of outer and inner claw (aot, ait)
height of heel of outer claw (hoh)
length of heel of outer and inner claw (loh, lih).

The toe angles and height of heel were measured in the parlour every eight weeks in the middle of each eight week period.

The toe lengths and heel lengths were recorded in a weigh crush every eight weeks at the beginning of each period and at the end of the final period; the figures used in the analyses for each period, were the means of the measurements at the beginning of the period and at the beginning of the subsequent period. Prior to measuring, the hooves were brushed and washed to remove any dirt. The length of heel (distance from the basis of the bulb to the distal edge of the bulb) was measured using a standard divider and a ruler. For measurement positions, see Figure 3.1. The The other hoof shape measurements were measured as described in Chapter 2.

Hoof hardness and hoof growth and wear measurements were recorded in the weigh crush, whilst the cow's hoof was hoisted vertically. A rope tied around the pastern and secured just below the dew claws was used to hoist the cow's hoof.

Hoof hardness measurements were taken on the right hind outer claw every eight weeks at the beginning of each period and at the end of the final period. The figures used in the analyses were calculated as above. A hand held, dial gauge Shore A meter (DIN 53 505, ISO/R 868, Bareiss), which measures the degree of indentation of a spring loaded probe was used. The Shore A meter was pressed against the hoof until the flat base made contact, and the degree of indentation was read from the dial, which was calibrated according to the Shore industrial scale of hardness. The sites of the measurements were as follows:

abaxial wall, toe region (AWT)
abaxial wall mid region (AWM)
sole, toe region (ST)
sole, mid region (SM)
heel bulb, centre (HBC).

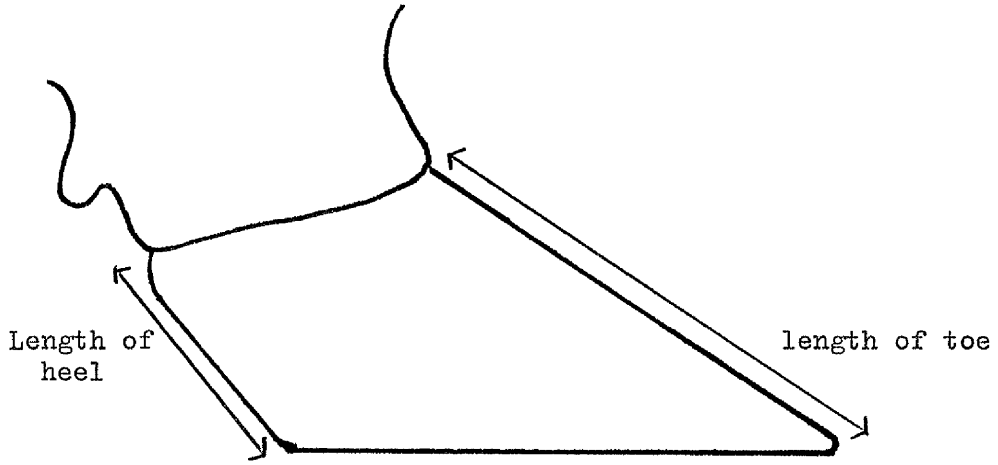
For site postitions, see Figure 3.2.

To estimate the repeatabilities of length of heel and hoof hardness measurements made on the same cow by the same observer, the 48 cows used in the experiment were recorded on 2 occasions separated by an interval of 60 minutes. The repeatabilities were calculated using the following equation:

$$r=var(cow)/\{var(cow)+var(record)+var(error)\}.$$

Hoof growth and wear measurements were taken in the dorsal

Figure 3.1 Hoof shape measurements



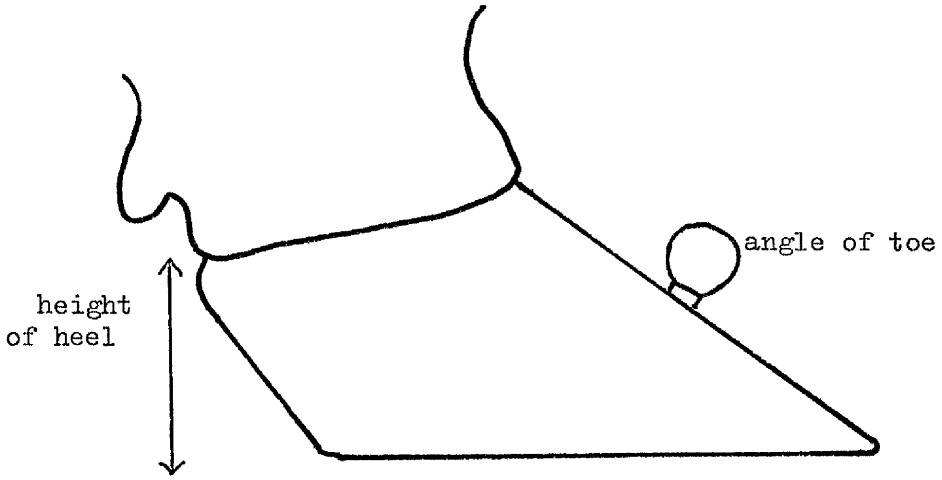
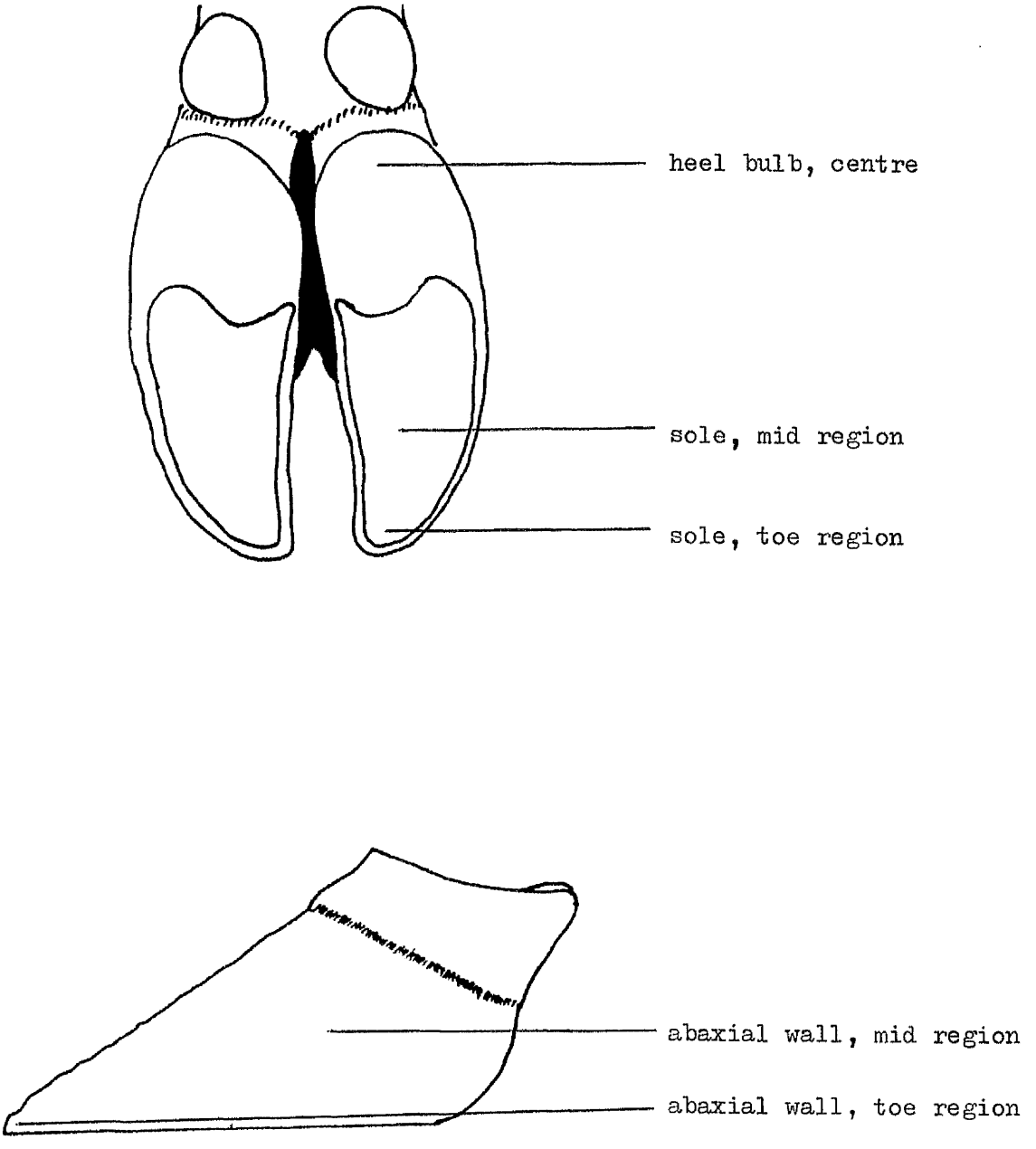


Figure 3.2 Hoof hardness measurements



region of the abaxial wall on the right hind outer claw. The procedure developed by Hahn (1979) was used. A mark which served as a reference point was burnt using a soldering iron (Weller S125D, Cooper Tools Ltd.), about 1 cm below the periople line. The distances from the top of the dorsal wall to the mark (A), and from the mark to the distal edge of the wall (B) were measured using conventional dividers, at the beginning of the first period. Subsequent measures were taken at the beginning of each period and at the end of the final period, and each time, a new mark was made about 1 cm below the periople line. The distances between the top of the dorsal wall and the new mark (C), the the old and the new mark (D), and the new mark and the distal edge (E) were recorded (see Figure 3.3). The monthly growth and wear rates were calculated using the following formulae: monthly

$$\text{monthly growth} = \frac{C+D-A}{\text{days between measurements}} \times 30.4$$

$$\text{monthly wear} = \frac{B-(E-D)}{\text{days between measurements}} \times 30.4$$

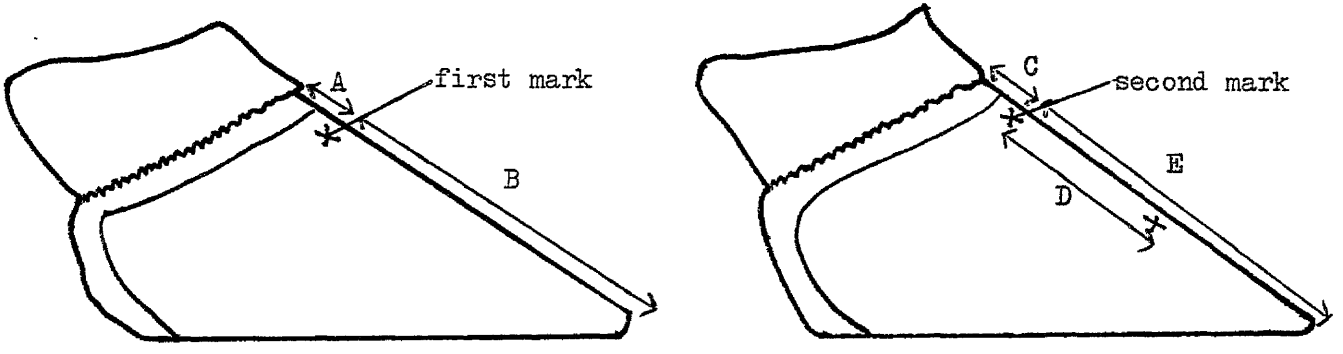
Blood samples

Blood samples were taken from the jugular vein using evacuated tubes and small bore (20G) needles at the beginning of each period and at the end of each period from each cow. The samples were analysed for blood Ca, Mg and P levels using a Multistart 3 microcentrifugal analyser, for CK levels using the Merck 1 test, NAC estimated, 30°C, and for serum GOT levels using the Merck 1 test, optimised, D6KC, 30°C. An average level of each variable obtained at the beginning and end of the period was used in the statistical analysis. Samples were also collected from animals who became clinically lame. The levels of the variables were correlated with locomotion score.

Statistical analysis

The results were analysed as a 2x2 factorial design (2 trimming treatments and 2 protein treatments) using the EDEX

Figure 3.3 Hoof growth and wear measurements



First measurement

Subsequent measurement

$$\text{Growth} = \frac{C+D-A}{\text{days between measurements}} \times 30.4$$

$$\text{Wear} = \frac{B-(E-D)}{\text{days between measurements}} \times 30.4$$

statistical package (Hunter, Patterson and Talbot, 1973, Edinburgh). Data for cows and heifers were analysed in successive three, eight week periods. A missing value was used for the second and third periods in the low protein, untrimmed treatment, as a cow died from a complicated mastitic condition. Minitab (Pennsylvania State University, 1980) was used to calculate correlation and regression coefficients between various measurements. As in the previous trial an analysis of variance was carried out to investigate whether it was necessary to adjust a current incident of lameness for a particular cow, for a previous incident of lameness, in the Chi-squared test, see Chapter 2. It was found that the adjustment using the covariate made no significant improvement to the analysis.

Data are presented as four, five week periods commencing at three weeks post calving.

RESULTS

Locomotion scores and clinical incidence of lameness

Trimming significantly reduced the locomotion scores in all three periods. The cows on the high protein diet had higher locomotion scores than those on the low protein diet, with significant differences being found in the third period and for the overall means. No interactions between protein level and trimming were found (see Table 3.14). There were indications that the differences in locomotion score between the high and low protein treatments increased as the trial progressed.

The number of clinical cases of lameness per cow week are shown in Table 3.15. Treatment was significantly ($p<0.001$) associated with the clinical incidence of lameness. Nearly two-thirds of all lameness cases were due to solar ulcers, and solar problems constituted 0.77 of all cases, see Table 3.16. Lameness cases caused by interdigital problems were minimal.

Correlation coefficients and regression equations for locomotion score on various hoof shape measurements are shown in Table 3.17.

Hoof growth and hoof wear

Higher growth rates and net growth rates were found for

Table 3.14 Locomotion score means

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 1	1.49b@	1.82ab	1.65ab	1.87a	0.12NS	0.12*	0.17NS
Period 2	1.38b	1.70ab	1.61ab	1.93a	0.13NS	0.13*	0.18NS
Period 3	1.33a	1.69b	1.65ab	1.95b	0.12*	0.12**	0.17NS
Means of periods 1 to 3	1.40a	1.74bc	1.64ab	1.92c	0.09*	0.09**	0.12NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.15 Incidence of lameness

	LT	LUT	HT	HUT
Number of clinical cases per cow week@	0.014	0.021	0.042	0.080

main treatment effects *** (Chi-squared analysis)

@ cows scoring 3 or more divided by the number of cow weeks (12x24)

Table 3.16 Diagnoses of clinical cases of lameness@

	Treatments			
	LT	LUT	HT	HUT
Number of examinations	4	6	12	23
Solar bruising	1	1	1	1
Solar ulcer	1	2	7	18
Solar penetration	1	2	0	0
White zone disease	1	0	0	0
Interdigital growth	0	0	0	1
Foot rot@	0	1	0	1
Leg injury	0	0	4	2

@ cows with locomotion scores of 3 or more

@ foot rot=dermatitis interdigitalis with underrunning

Table 3.17 Correlation coefficients and regression equations for locomotion score (Y) on hoof shape measurements (X).

Locomotion score (Y) on outer toe length (X) @	r=+0.604 (p<0.001)	y=-0.21+0.21(+0.04)X
Locomotion score (Y) on inner toe length (X) @	r=+0.619 (p<0.001)	y=-1.73+0.42(+0.08)X
Locomotion score (Y) on outer heel height (X) @	r=+0.111 (p>0.05)	y= 1.12+0.16(+0.21)X
Locomotion score (Y) on differences in claw length (X) @	r=+0.377 (p<0.001)	y= 1.27+0.17(+0.06)X
Locomotions score (Y) on inner heel length (X) (Period 1)	r=+0.361 (p<0.05)	y= 0.14+0.44(+0.17)X

@ values calculated using the means of periods 1 to 3

treatments LT and HT in periods 1 and 2 and for the overall means. This effect of trimming was significant on hoof growth in period 1 ($p<0.01$) and overall ($p<0.05$), and on net hoof growth in period 2 ($p<0.01$) and overall ($p<0.01$). Trimming also significantly ($p<0.05$) reduced hoof wear in period 2 (see Table 3.18).

The high protein level significantly ($p<0.01$) increased hoof growth in period 1, but no further significant effects were found (see Table 3.18).

There was a trend of decreasing hoof wear over the trial period, with the exception of treatment HT, which decreased and then increased.

Hoof growth exceeded hoof wear for all treatments and in all periods, with the exception of the low protein treatments in period 1 (see Table 3.18).

In the high protein group milk yield and protein yield were significantly correlated with hoof growth, see equations 3.1 and 3.2:

$r=-0.381$ ($p<0.05$)----------equation 3.1

$y=0.975-0.017(\pm 0.009)x$

where y=overall hoof growth and

x=overall milk yield

$r=-0.373$ ($P<0.05$)----------equation 3.2

$y=1.001-0.0005(\pm 0.0003)x$

where y=overall hoof growth and

x=overall protein yield

Hoof shape

Hoof shape means for periods 1 to 3 and for the overall means are shown in Table 3.19. The initial and final toe lengths and heel lengths are shown in Appendix 8. Trimming significantly reduced both outer and inner toe lengths in period 1 and overall, and its effects were significant ($p<0.01$) on the inner claw toe length in period 2. Trimming had no significant effect on toe

Table 3.18 Hoof growth and hoof wear means

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 1							
Hoof growth (cm/month)	0.52b@	0.41b	0.87a	0.57b	0.09**	0.09**	0.12NS
Hoof wear (cm/month)	0.58	0.72	0.53	0.50	0.14NS	0.14NS	0.19NS
Net hoof growth (cm/month)	-0.06b	-0.31b	0.34a	0.07ab	0.13**	0.13NS	0.18NS
Period 2							
Hoof growth (cm/month)	0.62	0.47	0.61	0.52	0.07NS	0.07NS	0.10NS
Hoof wear (cm/month)	0.27ab	0.43ab	0.24a	0.47b	0.09NS	0.09*	0.1! NS
Net hoof growth (cm/month)	0.35ab	0.04a	0.36b	0.06ab	0.11NS	0.11**	0.15NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 4.17 Hoof growth and hoof wear means

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 3							
Hoof growth (cm/month)	0.69a@	0.54b	0.59ab	0.46b	0.05NS	0.05*	0.07NS
Hoof wear (cm/month)	0.41	0.51	0.47	0.56	0.06NS	0.06NS	0.09NS
Net hoof growth (cm/month)	0.28a	0.03b	0.12ab	-0.10b	0.08NS	0.08**	0.12NS
Means of periods 1 to 3							
Hoof growth (cm/month)	0.60a	0.48bc	0.54ab	0.43c	0.03NS	0.03**	0.05NS
Hoof wear (cm/month)	0.43	0.47	0.38	0.47	0.04NS	0.04NS	0.06NS
Net hoof growth (cm/month)	0.17a	0.01b	0.16a	-0.04b	0.05NS	0.05**	0.08NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.19 Hoof shape means

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 1							
Length of toe (outer) (cm)	7.92a@	9.10b	8.43b	9.37b	0.20NS	0.20***	0.28NS
Length of toe (inner) (cm)	7.76a	8.62b	7.88a	8.35b	0.12NS	0.12***	0.18NS
Angle of toe (outer) (o)	49.1a	40.9b	46.1ab	40.6b	2.03NS	2.03**	2.87NS
Angle of toe (inner) (o)	49.9a	43.1b	49.4ac	45.1bc	1.56NS	1.56**	2.21NS
Height of heel (outer) (cm)	3.14	3.29	3.36	3.23	0.13NS	0.13NS	0.19NS
Length of heel (outer) (cm)	3.64	3.91	3.74	3.70	0.11NS	0.11NS	0.16NS
Length of heel (inner) (cm)	3.50	3.80	3.51	3.52	0.10NS	0.10NS	0.15NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.19 Hoof shape means

(continued)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 2							
Length of toe (outer) (cm)	8.30a@	8.83ab	9.06ab	9.50b	0.28*	0.28NS	0.40NS
Length of toe (inner) (cm)	7.93b	8.47a	8.00b	8.25ab	0.14NS	0.14**	0.19NS
Angle of toe (outer) (o)	41.6	33.8	37.5	38.5	3.28NS	3.28NS	4.64NS
Angle of toe (inner) (o)	45.0	41.9	45.3	44.9	1.66NS	1.66NS	2.35NS
Height of heel (outer) (cm)	3.48	3.55	3.43	3.29	0.12NS	0.12NS	0.17NS
Length of heel (outer) (cm)	3.69	3.86	3.74	3.56	0.10NS	0.10NS	0.15NS
Length of heel (inner) (cm)	3.56	3.58	3.48	3.37	0.10NS	0.10NS	0.15

@ means with different subscripts are significantly different

Table 3.19 Hoof shape means

(continued)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 3							
Length of toe (outer) (cm)	8.91	9.11	9.50	9.68	0.32NS	0.32NS	0.45NS
Length of toe (inner) (cm)	8.22	8.31	8.20	8.46	0.13NS	0.13NS	0.19NS
Angle of toe (outer) (o)	36.7	31.8	31.4	40.9	3.56NS	3.56NS	5.04NS
Angle of toe (inner) (o)	45.3	42.7	44.3	44.9	1.41NS	1.41NS	1.99NS
Height of heel (outer) (cm)	3.46	3.59	3.66	3.43	0.10NS	0.10NS	0.14NS
Length of heel (outer) (cm)	3.74ab@	4.01a	3.97ab	3.70b	0.11NS	0.11NS	0.15*
Length of heel (inner) (cm)	3.61	3.73	3.68	3.54	0.10NS	0.10NS	0.14NS

@ means with different subscripts are significantly different

Table 3.19 Hoof shape means

(continued)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Means of periods 1 to 3							
Length of toe (outer) (cm)	8.38a@	9.01ab	8.99ab	9.52b	0.25*	0.25*	0.35NS
Length of toe (inner) (cm)	7.97a	8.44b	8.03ac	8.35b	0.11NS	0.11***	0.15NS
Angle of toe (outer) (o)	42.3	36.0	36.6	40.0	2.54NS	2.54NS	3.59NS
Angle of toe (inner) (o)	46.8a	43.0b	46.3ab	44.8ab	1.18NS	1.18*	1.67NS
Height of heel (outer) (cm)	3.37	3.49	3.49	3.32	0.09NS	0.09NS	0.12NS
Length of heel (outer) (cm)	3.69	3.93	3.79	3.65	0.09NS	0.09NS	0.14NS
Length of heel (inner) (cm)	3.55	3.71	3.55	3.48	0.08NS	0.08NS	0.12NS

@ means with different subscripts are significantly different

lengths in period 3. The high protein level significantly ($p<0.05$) increased the outer claw toe length in period 2 and overall, but there were no other significant effects of protein on toe length.

Toe angles were significantly ($p<0.01$) steeper on the trimmed treatments in period 1 for both outer and inner claws. Trimming also significantly ($p<0.05$) increased inner claw angle overall. Protein level had no significant effects on toe angle.

No significant effects of either trimming or protein level on the the heel height of the outer claw in any period were found.

There was a significant ($p<0.05$) interaction between trimming and protein level on the outer claw heel length in period 3, which indicated that cows on treatment LUT had significantly longer heels than those on treatment HUT. No other treatment effects on heel length were found.

A trend of increasing outer claw toe length over the trial period, with the exception of treatment LUT, was found. There was also a trend of decreasing outer toe angle, with the exception of treatment HUT. As the trial progressed a gradual convergence between the treatments of inner claw toe length was found.

The correlation coefficients and regression equations between angle of toe and length of toe are shown in Table 3.20.

The within observer repeatabilities of length of outer heel and length of inner heel were 0.84 and 0.83, respectively.

Hoof hardness

The means of the initial and final hoof hardness measuremets are shown in Appendix 9.

No treatment effects on abaxial wall toe, abaxial wall mid, sole toe or sole mid hardness were found. However, trimming significantly reduced heel bulb centre hardness in period 1

Table 3.20 Correlation coefficients and regression equations for angle of toe (Y) on length of toe (X)

	Outer claw	Inner claw
Angle of toe (Y) on	r=-0.707 (p<0.001)	r=-0.448 (p<0.01)
length of toe (X) period 1	y=102.3-6.7(±1.00)X	y=91.0-5.5(±1.62)X
Angle of toe (Y) on	r=-0.651 (p<0.001)	r=-0.419 (p<0.01)
length of toe (X) period 2	y=104.6-7.5(±1.30)X	y=83.5-4.8(±1.55)X
Angle of toe (Y) on	r=0.692 (p<0.001)	r=-0.375 (p<0.01)
length of toe (X) period 3	y=108.5-7.9(±1.22)X	y=75.0-3.7(±1.36)X
Angle of toe (Y) on	r=-0.755 (p<0.001)	r=-0.472 (p<0.001)
length of toe (X)	y=104.0-7.2(±0.94)X	y=83.2-4.7(±1.29)X
mean of periods 1 to 3		

($p<0.05$), period 2 ($p<0.01$) and overall ($p<0.05$) (see Table 3.21).

The overall abaxial wall toe region was harder than the abaxial wall mid region, except on treatment LT, and the abaxial wall mid region was in turn harder than the sole toe region. The sole toe region was harder than the sole mid region. The softest region was the heel bulb centre. A significant correlation between the overall sole mid hardness and overall locomotion score was found, see equation 3.3.

$r=-0.333$ ($p<0.05$)----------equation 3.3

$y=3.64-0.03(\pm 0.01)x$

where y=overall locomotion score and

x=overall sole mid hardness

No particular trends of hardness with stage of lactation were found.

The within observer repeatabilities of hoof hardness measurements were as follows: abaxial wall toe , 0.80; abaxial wall mid , 0.79; sole toe , 0.81; sole mid , 0.78 and heel bulb centre , 0.72.

Cow performance and blood analyses

The mean daily intakes of dry matter and calculated ME, RDP and UDP intakes are shown in Table 3.22.

The analyses of the production parameters were adjusted using covariates based on the 14 day post-partum values, except for the liveweight change and condition score change analyses which were analysed using unadjusted values.

The high protein diet significantly ($p<0.05$) increased milk yield in period 2 and overall. Milk fat contents were significantly ($p<0.05$) less in period 2, and milk lactose contents were significantly ($p<0.05$) more overall on the high protein treatments. There were no significant effects of protein level on

Table 3.21 Hoof hardness means (Shore A units, scale 0 to 100)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 1							
Abaxial wall toe	88.0	88.1	89.9	87.7	1.94NS	1.94NS	2.74NS
Abaxial wall mid	88.8	85.1	86.5	84.2	1.68NS	1.68NS	2.38NS
Sole toe	82.9	82.4	80.8	78.3	2.07NS	2.07NS	2.93NS
Sole mid	74.5	68.0	72.7	70.2	2.33NS	2.33NS	3.30NS
Heel bulb centre	36.3ab@	42.5a	35.6b	39.6ab	2.25NS	2.25*	3.18NS
Period 2							
Abaxial wall toe	90.2	91.6	93.1	91.9	1.03NS	1.03NS	1.46NS
Abaxial wall mid	90.2	89.7	90.3	89.7	1.14NS	1.14NS	1.62NS
Sole toe	87.1	88.4	86.3	86.2	1.42NS	1.42NS	2.00NS
Sole mid	77.7	76.0	77.0	76.2	1.53NS	1.53NS	2.17NS
Heel bulb centre	38.3ab	43.3ab	36.5a	44.6b	2.33NS	2.33**	3.30NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.21 Hoof hardness means (Shore A units, scale 0 to 100)
(continued)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 3							
Abaxial wall toe	88.4	88.5	88.1	87.9	1.08NS	1.08NS	1.52NS
Abaxial wall mid	88.3	87.7	87.6	87.8	1.02NS	1.02NS	1.44NS
Sole toe	86.6	86.5	85.7	85.3	1.01NS	1.01NS	1.43NS
Sole mid	78.5	78.3	75.6	76.2	1.37NS	1.37NS	1.94NS
Heel bulb centre	39.0	40.6	38.8	44.8	2.38NS	2.38NS	3.36NS
Means of periods 1 to 3							
Abaxial wall toe	88.8	89.4	90.4	89.2	0.98NS	0.98NS	1.39NS
Abaxial wall mid	89.1	87.5	88.1	87.2	1.04NS	1.04NS	1.47NS
Sole toe	85.5	85.8	84.3	83.3	1.05NS	1.05NS	1.48NS
Sole mid	77.2	73.8	75.1	74.2	1.23NS	1.23NS	1.74NS
Heel bulb centre	37.9abc	42.1ab	37.0a	43.0b	1.91NS	1.91*	2.71NS

@ means with different subscripts are significantly different ($p<0.05$)

milk protein content, but milk protein yields were significantly ($p<0.01$) higher on the high protein treatments in period 2. No significant effects of protein level on fat yield were found. There were no significant effects of trimming on any of the milk production parameters (see Table 3.23).

The cows on the low protein diet were significantly heavier in periods 2 and 3, and overall. No effects of trimming on liveweight or liveweight change were found (see Table 3.24). The condition scores were significantly ($p<0.05$) lower on the high protein, untrimmed treatment. However, there were no significant differences in condition score change between the treatments (see Table 3.25).

Serum GOT levels were significantly higher on the high protein treatments in all periods. CK and P levels were also significantly higher on the high protein treatments in periods 2 and 3 (see Table 3.26).

There was a significant, postitive correlation between Mg level and sole mid hardness ($r=+0.362$, $p<0.05$) in period 1, and between Ca level and sole toe hardness ($r=+0.332$, $p<0.05$) in period 3. No significant correlations between locomotion score and the blood parameters were found.

Table 3.22 Mean daily intakes of dry matter and the calculated ME, CP, RDP, and UDP intakes

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 1							
Complete diet DM intake (kg/day)	14.7	15.5	15.0	14.6	0.44NS	0.44NS	0.62NS
Concentrate DM intake (kg/day)	1.7	1.7	1.7	1.7			
Total DM intake (kg/day)	16.4	17.2	16.7	16.3	0.44NS	0.44NS	0.62NS
Total ME intake (MJ/day)	200.3	209.3	205.3	199.9	5.31NS	5.31NS	7.52NS
Total CP intake (g/day)	2565a@	2668a	3341b	3251b	77.36***	77.36NS	109.40NS
Total RDP intake (g/day)	1997a	2086a	2562b	2492b	60.24***	60.24NS	85.19NS
Total UDP intake (g/day)	568a	582a	779b	759b	17.69***	17.69NS	25.02NS
Conc:forage ratio	61:39		61:39				

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.22 Mean daily intakes of dry matter and the calculated ME, CP, RDP, and UDP intakes (continued)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 2							
Complete diet DM intake (kg/day)	14.6	15.0	14.9	14.3	0.34NS	0.34NS	0.49NS
Concentrate DM intake (kg/day)	1.7	1.7	1.7	1.7			
Total DM intake (kg/day)	16.3	16.7	16.6	16.0	0.34NS	0.34NS	0.49NS
Total ME intake (MJ/day)	201.6	206.5	204.3	197.2	4.18NS	4.18NS	5.92NS
Total CP intake (g/day)	2529a@	2586a	3284b	3165b	63.27***	63.27NS	89.47NS
Total RDP intake (g/day)	1956a	2001a	2507b	2417b	49.5***	49.5NS	69.95NS
Total UDP intake (g/day)	573a	585a	777b	748b	13.97***	13.97NS	19.76NS
Conc:forage ratio	59:41		59:41				

@ means with different subscripts are significantly different

Table 3.22 Mean daily intakes of dry matter and the calculated ME, CP, RDP, and UDP intakes

(continued)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 3							
Complete diet DM intake (kg/day)	14.6	14.5	14.5	14.2	0.36NS	0.36NS	0.51NS
Concentrate DM intake (kg/day)	1.7	1.7	1.7	1.7			
Total DM intake (kg/day)	16.3	16.2	16.2	15.9	0.36NS	0.36NS	0.51NS
Total ME intake (MJ/day)	196.6	196.1	196.2	192.8	4.30NS	4.30NS	6.08NS
Total CP intake (g/day)	2774a@	2768a	3261b	3202b	70.53***	70.53NS	99.75NS
Total RDP intake (g/day)	2163a	2154a	2513b	2467b	55.45***	55.45NS	78.42NS
Total UDP intake (g/day)	611a	614a	748b	735b	15.30***	15.30NS	21.67NS
Conc:forage ratio	60:40		60:40				

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.22 Mean daily intakes of dry matter and the calculated ME, CP, RDP, and UDP intakes

(continued)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Mean of periods 1 to 3							
Complete diet DM intake (kg/day)	14.6	15.0	14.8	14.4	0.28NS	0.28NS	0.39NS
Concentrate DM intake (kg/day)	1.7	1.7	1.7	1.7			
Total DM intake (kg/day)	16.3	16.7	16.5	16.1	0.28NS	0.28NS	0.39NS
Total ME intake (MJ/day)	195.5	204.0	201.9	196.6	3.34NS	3.34NS	4.72NS
Total CP intake (g/day)	2623a@	2674a	3295b	3206b	51.64***	51.64NS	73.00NS
Total RDP intake (g/day)	2039a	2080a	2527b	2459b	40.23***	40.23NS	56.89NS
Total UDP intake (g/day)	584a	594a	768b	747b	11.60***	11.60NS	16.40NS
Conc:forage ratio	60:40		60:40				

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.23 Milk yield and milk compos ition means

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 1							
Milk yield (kg/day)	24.0	24.6	25.6	26.1	0.78NS	0.78NS	1.11NS
Milk fat content (g/kg)	40.7	41.0	40.3	41.2	0.77NS	0.77NS	1.09NS
Milk fat yield (g/day)	970	1013	1027	1060	30.5NS	30.5NS	45.1NS
Milk protein content (g/kg)	33.9	34.2	34.0	33.5	0.46NS	0.46NS	0.65NS
Milk protein yield (g/day)	804	853	863	874	28.0NS	28.0NS	39.6NS
Period 2							
Milk yield (kg/day)	21.6a@	21.9ab	23.7ab	24.2b	0.80*	0.80NS	1.13NS
Milk fat content (g/kg)	45.4ab	46.4a	43.2ab	43.0b	1.17*	1.17NS	1.63NS
Milk fat yield (g/day)	966	1015	1019	1029	33.0NS	33.0NS	46.6NS
Milk protein content (g/kg)	35.8	36.5	36.1	35.7	0.60NS	0.60NS	0.85NS
Milk protein yield (g/day)	768a	805ab	849b	860b	25.3**	25.3NS	35.8NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.23 Milk yield and milk compos ition means

(continued)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 3							
Milk yield (kg/day)	20.5	20.5	21.6	21.7	0.90NS	0.90NS	1.23NS
Milk fat content (g/kg)	46.9	45.7	44.0	43.7	1.29NS	1.29NS	1.82NS
Milk fat yield (g/day)	953	930	942	939	32.7NS	32.7NS	46.3NS
Milk protein content (g/kg)	36.8	37.3	36.5	36.2	0.62NS	0.62NS	0.87NS
Milk protein yield (g/day)	746	766	781	784	26.6NS	26.6NS	37.7NS
Means of periods 1 to 3							
Milk yield (kg/day)	21.9a@	22.3ab	23.6ab	24.0b	0.78*	0.78NS	1.00NS
Milk fat content (g/kg)	44.3	44.3	42.5	42.6	1.00NS	1.00NS	1.41NS
Milk fat yield (g/day)	962	986	996	1009	29.4NS	29.4NS	41.6NS
Milk protein content (g/kg)	35.5	36.0	35.5	35.1	0.52NS	0.52NS	0.73NS
Milk protein yield (g/day)	773	808	831	839	24.1NS	24.1NS	34.0NS
Milk lactose content (g/kg)	47.9ab	47.8a	48.6b	48.3ab	0.25*	0.25NS	0.36NS
Milk s-n-f content (g/kg)	90.8	91.1	91.5	90.9	0.72NS	0.72NS	1.02NS
Milk total solids (g/kg)	134.8	135.4	134.0	133.4	1.37NS	1.37NS	1.93NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.24 Liveweight and liveweight change means

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim (T)	PxT
Period 1							
Liveweight (kg)	554	553	549	534	7.08NS	7.05NS	9.94NS
Liveweight change (kg/day)	+0.24	+0.38	+0.01	-0.06	0.19NS	0.19NS	0.26NS
Period 2							
Liveweight (kg)	572a	577a	559ab	540b	7.14**	7.14NS	10.10NS
Liveweight change (kg/day)	+0.27	+0.26	+0.29	+0.15	0.07NS	0.07NS	0.10NS
Period 3							
Liveweight (kg)	595a	596a	580a	552b	7.43***	7.43NS	10.50NS
Liveweight change (kg/day)	+0.27	+0.39	+0.44	+0.29	0.07NS	0.07NS	0.10NS
Means of periods 1 to 3							
Liveweight (kg)	574a	575a	563a	542b	6.05***	6.05NS	8.55NS
Liveweight change (kg/day)	+0.26	+0.33	+0.24	+0.13	0.07NS	0.07NS	0.10NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.25 Condition score and condition score change means

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Period 1							
Condition score	2.22	2.04	2.12	1.98	0.10NS	0.10NS	0.13NS
Condition score change	-0.003	-0.043	-0.014	-0.003	0.02NS	0.02NS	0.03NS
Period 2							
Condition score	2.31a@	2.22a	2.18a	1.79b	0.11*	0.11*	0.16NS
Condition score change	-0.103	-0.112	-0.099	-0.090	0.02NS	0.02NS	0.03NS
Period 3							
Condition score	2.53a	2.43a	2.47a	1.98b	0.11*	0.11*	0.15NS
Condition socre change	+0.139	+0.143	+0.149	+0.127	0.02NS	0.02NS	0.03NS
Means of periods 1 to 3							
Condition score	2.35a	2.23a	2.26a	1.92b	0.10*	0.10*	0.14NS
Condition score change	+0.011	-0.004	+0.012	+0.001	0.01NS	0.01NS	0.02NS

@ means with different subscripts are significantly different

Table 3.26 Blood parameter level means

	Treatments				SED		
	LT	LUT	HT	HUT	protein (P)	trim(T)	PxT
Period 1							
Ca (mmol/l)	2.34	2.36	2.26	2.32	0.004NS	0.004NS	0.063NS
Mg (mmol/l)	1.19	1.14	1.14	1.16	0.026NS	0.026NS	0.037NS
P (mmol/l)	1.73	1.64	1.84	1.82	0.071NS	0.071NS	0.100NS
sGOT, 38oC (iu/l)	43.3b@	43.2b	52.4a	49.2ab	2.28***	2.28NS	3.22NS
CK, 25oC (iu/l)	36.8	31.4	45.0	41.5	4.86NS	4.86NS	6.87NS
Period 2							
Ca (mmol/l)	2.38a	2.53b	2.37a	2.46ab	0.042NS	0.042**	0.059NS
Mg (mmol/l)	1.16	1.15	1.11	1.14	0.028NS	0.028NS	0.039NS
P (mmol/l)	1.80ab	1.75a	1.93b	1.85ab	0.046*	0.046NS	0.065NS
sGOT, 38oC (iu/l)	57.1a	61.6a	80.4b	76.8b	4.60***	4.60NS	6.50NS
CK, 25oC (iu/l)	43.3a	45.5ab	66.0b	66.1b	7.44**	7.44NS	10.52NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 3.26 Blood parameter level means

(continued)

	Treatments				SED		
	LT	LUT	HT	HUT	protein (P)	trim(T)	PxT
Period 3							
Ca (mmol/l)	2.44	2.52	2.44	2.49	0.031NS	0.031NS	0.043NS
Mg (mmol/l)	1.10	1.10	1.08	1.10	0.016NS	0.016NS	0.022NS
P (mmol/l)	1.87ab@	1.80a	1.99b	1.89ab	0.044*	0.044NS	0.062NS
sGOT, 38oC (iu/l)	69.7a	72.2ac	94.0b	90.8bc	6.77**	6.77NS	9.57NS
CK, 25oC (iu/l)	44.0a	45.3a	56.6ab	68.0b	7.39*	7.39NS	10.45NS
Means of periods 1 to 3							
Ca (mmol/l)	2.39	2.46	2.37	2.41	0.031NS	0.031NS	0.045NS
Mg (mmol/l)	1.15	1.13	1.11	1.13	0.018NS	0.018NS	0.026NS
P (mmol/l)	1.80ab	1.72a	1.92b	1.85ab	0.047*	0.047NS	0.067NS
sGOT, 38oC (iu/l)	56.6a	58.0a	75.7b	71.4b	3.88***	3.88NS	5.49NS
CK, 25oC (iu/l)	41.0ac	39.1a	54.3bc	57.2b	4.28**	4.28NS	6.05NS

@ means with different subscripts are significantly different ($p<0.05$)

DISCUSSION

Locomotion scores and clinical incidence of lameness

The finding that trimming significantly improved locomotion and reduced the clinical incidence of lameness is in contrast to the results of Arkins (1981), which showed that trimming did not significantly reduce the incidence of digital disease. However, the results from this experiment concur with the suggestions of several authors (Sandelien, 1960; Smedegaard, 1963; Weaver, 1979; Edwards, 1980) that trimming is effective in preventing lameness.

The poorer locomotion scores of the cows with untrimmed hooves were probably due to unequal weight bearing between the outer and inner claws and poor weight distribution within the claws. Large differences between the outer and inner claw toe lengths were associated with higher locomotion scores ($r=+0.377$, $p<0.01$), and longer toe lengths of both the outer and inner claws were also associated with poorer locomotion, ($r=+0.604$, $p<0.001$) and ($r=+0.619$, $p<0.001$) respectively. Undue mechanical pressure on the solar corium, which occurs when too much weight is borne by the pedal bone or when the corium is inadequately supported by the horny sole, is thought to predispose to solar contusions Toussaint Raven, 1973). Such a situation would have existed in the claws of the untrimmed treatments. Higher heels have also been associated with solar contusions. However, in this trial there were no significant correlations between height of heel and locomotion score ($r=+0.111$, NS), which indicated that toe length and differences in toe length between the claws, were probably more important than heel height in predisposing to hoof problems.

Thrombosis formation and loss of corial capillary integrity could be implicated in the pathogenic effects of the high protein level on laminitis. Nilsson (1963; 1966) suggested that thromboses developed as allergic-histaminotic reactions in response to the feeding of high levels of protein rich concentrates. Thromboses would prevent adequate supplies of sulphur amino acids from reaching the keratin producing cells. The high protein diets may also have led to the production

of toxins of proteinaceous origin. Such toxins have been associated with a drain on chondrotin sulphate of horn tissue (Urmas, 1965; Chew, 1972) and the partial blocking of cystine metabolism in the hoof matrix (Larsson et al, 1956). They are also thought to prevent the transfer of amino acids from the corial capillaries to the keratin producing cells through a toxic influence on the capillary walls (Edwards, 1982). These factors would lead to the production of poor quality horn tissue. In turn this would predispose to hoof problems and therefore higher locomotion scores, as were found in the high protein treatments.

Additionally, the high protein levels may have worsened locomotion in the first period through their effects on hoof growth. The significantly ($p<0.05$) longer outer claws in the high protein treatments in period 2, which would cause poor weight distribution, were partly due to the significantly ($p<0.05$) higher growth rates in period 1.

The gradual effects of the toxins on the capillary walls and the time that the poorly formed horn cells would have taken to reach the distal surface of the hoof, may have explained the increasing divergence between the low and high protein treatments in locomotion score over the trial period. As the malformed cells reached the surface the hoof would have become increasingly predisposed to solar problems, for example solar ulceration.

In treatment HUT the majority of clinical cases of lameness were due to solar problems, and this in conjunction with the mean locomotion score, appears to be indicative of a laminitis-solar ulcer type syndrome existing in this treatment. The onset of laminitis is associated with tenderness and uneveness of gait, which are reflected in mean scores of about 2. The laminitic condition is related to hoof problems as described above.

Hoof growth and hoof wear

The positive effect of trimming on hoof growth may have been due to some compensatory mechanism, as yet unspecified. However, Hamilton et al (1955) have suggested that increased nail growth,

associated with nail-biting, was comparable to increased proliferative rates obtained in other circumstances when cells were jostled.

Rates of hoof growth may also be influenced both by levels of milk production, in particular milk protein yields, and laminitic factors. Hahn (1979) has suggested that in high yielding dairy cows the milk producing tissues compete with the horn building tissues for protein components. Dietz and Koch (1972) also found that high producing cows had lower rates of hoof growth. Similarly, in this trial negative correlations were found between rate of hoof growth and milk yield, and between rate of hoof growth and protein yield in the high protein group.

Laminitis may also reduce potential rates of growth. The effects of laminitis, for example, capillary damage and stagnation of blood in the corial capillaries, may cause an insufficient nutrient supply to the underlying horn producing cells. However, long toes have been associated with laminitis (Greenough et al, 1981), but no surveys or trials have been conducted to relate toe length with severity or type of laminitis (acute or chronic). It is possible that the long toes may only result when laminitis is chronic over a long period of time. In the herd from which the cows were taken, trimming was carried out annually and where necessary, and so any long term effects of chronic laminitis would have been masked to some extent. The trimming would also have reduced the effects of chronic laminitis on function and shape; the use of trimming as a curative measure for laminitis has been recommended by Toussaint-Raven (1971). A better knowledge of the relationship between hoof growth and laminitis is also required. How extensively a corial disorder in one part of the hoof affects growth in another part is little understood. Toussaint Raven (1985) has suggested that the corium reacts to a disorder in the germinal layer by means of a better blood circulation. Therefore, horn formation may stagnate in one area because of a localised disorder in the germinal layer, but at the same time may be stimulated in the rest of the claw by the general reaction of the corium. However, there is no evidence for this and further research in this area is required.

In the first period protein yields were similar in both low

and high protein treatments and so the competition factor would have been of the same relative importance for both groups. Therefore, there would have been relatively more protein available for hoof growth for the cows on the high protein diets, as was indicated by the significantly higher growth rates. There were no significant effects of protein level on locomotion score in this period, and therefore it would have been expected that differences in levels of laminitis between the groups would be minimal. Therefore, the influence of the laminitic factor on the determination of levels of hoof growth would have been similar in both the low and high protein groups.

Protein yields were significantly higher in the high protein group in period 2. Therefore, the competition factor would have been relatively more important for the cows on the high protein treatments, and consequently relatively less protein would have been available for hoof growth. The differences in the levels and severity of the physiological and pathological effects of laminitis were probably similar in both groups, since the differences between the groups in locomotion score were similar Therefore, the rates of hoof growth in the low and high protein treatments would have been more alike in period 2 than in period 1, since the growth rates of the high protein treatments were lower in period 2.

In the third period protein yields were similar in both groups, but locomotion scores were significantly higher in the high protein group, which probably indicated that some of the effects of laminitis, for example, capillary damage and thromboses formation, had become marked and persistent. Therefore, the potential for hoof growth on the high protein treatment would have been reduced, and so the effects of the higher levels of dietary protein which were available for hoof growth, would have been over-ridden.

The finding that hoof wear decreased over the trial period corresponds to the observation of Hahn (1979), who found negative regression values for rate of hoof wear on stage of lactation in first lactation cows. He suggested that this indicated that a

change of environment from pasture, where the heifers were previously kept, to concrete produced a higher rate of wear at the beginning of lactation. This coincides with the management of the cows and heifers on this trial.

Hoof shape

Toe length is determined both by trimming and by hoof growth and wear. The differences between the trimmed and the untrimmed outer claw toe lengths of the cows in the high protein group decreased over the trial period. This was because the rate of hoof growth of the trimmed treatment was greater, and the rate of hoof wear was smaller than that of the untrimmed treatment in all periods. However, in the low protein group these differences in toe length between the trimmed and untrimmed treatments decreased from the start of the trial to the third period and then increased from the third period to the end of the trial. Again these changes corresponded to the differences between the trimmed and untrimmed treatments in rates of growth and wear. Net growth (growth minus wear) was greater in the trimmed treatment in periods 1 and 2, whereas in period 3 net growth was slightly more in the untrimmed treatment.

By stimulating hoof growth and reducing hoof wear the initial effects of trimming in reducing outer toe length were lessened, and became non significant at about seven months. (Trimming was carried out about four months before the trial started, and the toe length measurements for period 2, shown in Table 3.19, are the means of toe lengths recorded at 2 and 4 months after the start of the trial). Therefore, the benefit of trimming in reducing hoof problems through its effect on decreasing toe length, (toe length was negatively correlated with locomotion score), tended to disappear about seven months after trimming. However, trimming significantly reduced locomotion score in all three periods and this was probably due to its additional effects on other aspects of hoof shape and function. These aspects included the minimizing of differences between the outer and inner claws in toe length and heel height (differences

in toe length between the claws were negatively correlated with locomotion score), shaping of the axial borders so that slurry was drained away from the hoof and curative trimming. Curative trimming comprises the removal of loose and necrotic horn, trimming of the horny borders which exert pressure and the following out of cracks. Therefore, whilst the results indicate that annual trimming improves locomotion and reduces the incidence of lameness to a very acceptable level (the time from trimming to the end of the trial was 10 months), biannual trimming may be of considerable benefit since toe lengths would remain reasonably short throughout the year. However, before a conclusive assessment of the merits of biannual trimming compared to once yearly trimming can be made, the changes with time of the other aspects of hoof structure and function, which trimming alters, need to be studied. The relative importance of each aspect, for example, cleft model and toe length, with respect to locomotion, would then need to be investigated.

As discussed previously, locomotion score was associated with toe length and the differences between the outer and inner claws in toe length.

The effect of the high protein level in increasing outer claw toe length in period 2 and overall, was due to the high protein level increasing hoof growth in the first period.

Overall angle of toe was negatively correlated with overall toe length ($r=-0.755$, $p<0.001$ for outer toe; $r=-0.472$, $p<0.001$ for inner toe). However, the use of length of toe as a predictor of angle of toe should be used with caution, since correlations significant at the 0.1% level were not consistently found.

The effect of non trimming on increasing heel length appeared to be reduced by the high protein level. The high protein level may have increased the vulnerability of the distal, posterior surface of the heel to abrasion and wear by predisposing to laminitis, and therefore to production of faulty horn. Increased abrasion and wear would then have reduced the heel length. However, to what extent the corium of the heel

region is affected by laminitis is unclear, although blood staining of the sole-heel junction has been attributed to primary acute laminitis (Edwards, 1982).

Although there was a significant correlation ($r=+0.361$, $p<0.05$) between locomotion score and inner heel length in period 1, no further indications of an association between heel length or heel height and locomotion score were found. These results are in contrast to the suggestion that higher heels result in sensitivity on standing and abduction of the hind legs (Toussaint Raven, 1973). (The degree of abduction is incorporated into the locomotion scoring system). However, the absence of a correlation in this trial may have been due to the small variation between cows in both heel height and heel length.

It may be concluded from the repeatabilities of the heel length measurements, that heel length can be recorded with reasonable precision.

Hoof hardness

The main factors determining hardness are probably moisture content and amino acid composition of the horn. Over the trial period the cumulative effects of the hooves being in contact with slurry so elevating moisture contents of horn, would be expected to result in increasing softness. However, no such trend was found, and this may have been due to the decline in milk protein yield over the trial period. The milk producing tissues are thought to compete with the horn producing tissues for protein components (Hahn, 1979), and so such a decline in milk protein yield may have increased the availability of sulphur amino acids for disulphide bonding in horn production. Harder keratin tissue has been attributed to higher levels of disulphide bonding (Clark and Rakes, 1982). It is uncertain as to which amino acid there is most competition for. However, methionine may be important, since several researchers have found increased wool growth when methionine or methionine hydroxy analog was infused into the abomasum (Reis, 1967; Wright, 1969; Bird and Moir, 1972). Fisher (1972) and Schwab and Satter (1974) also reported

that methionine was limiting for milk protein synthesis. Thus, the increased availability of amino acids may have counterbalanced the cumulative effects of hoof contact with slurry. However, no firm conclusions can be drawn about milk protein yield changes in relation to concurrent reponses in hoof hardness, since dietary protein level as a measure of the supply of amino acids to the tissues is inadequate.

Less weight bearing by the heel, with subsequently lower levels of compression of the fibro-elastic tissue of the bulb, may have accounted for the effect of trimming on reducing heel bulb hardness. A soft heel bulb is advantageous since its function is to act as a cushion so spreading the load evenly, minimizing internal stresses and absorbing strain energy (Webb et al, 1984).

The negative correlation between mid sole hardness and locomotion score probably reflects the enhanced ability of a harder sole to provide support and protection for the corium. Lower locomotion scores are likely to be associated with a more healthy corium.

Trimming, by improving hoof shape and allowing better drainage of slurry from the solar axial region, might be expected to reduce moisture contents and maceration of the horn, and therefore to increase the hardness of the two solar sites measured. Hydrated horn has been associated with softer horn (Fritsch, 1966; Prentice, 1972). However, no such relationship between trimming and hardness was found. Low levels of slurry in the housing area due to twice daily scraping of the passageways may have accounted for the lack of a relationship.

The repeatabilities of the hoof hardness measurements indicated that hoof hardness could be measured with a reasonable degree of precision using the Shore A meter. The lower repeatability of the heel bulb centre was probably due to the surface of the bulb being rutted and uneven in some of the cows. To give reproducible results the test surface should be flat or convex over an area the size of the base of the meter.

Cow performance

The results of this trial can not strictly be compared with other trials which have examined the effects of protein level on cow performance, because many have confounded CP intake with DM and ME intake. In this trial mean ME intakes were maintained at the same level for all treatments.

The milk yield results were in conflict with those of Barney et al (1981) and Ha and Kennelly (1984), who found no significant differences in the milk yields of cows when protein rations were increased from 12% to 18% CP, and from 13% to 19% CP respectively. However, the lack of a treatment effect on protein content agreed with the results of Majdoub et al (1977), but conflicted with the findings of other authors who found trends of increasing milk protein content with increasing CP intakes (Barney et al, 1981; Holter et al, 1982).

The significantly higher fat content found on the 16% CP treatment was in accordance with the suggestion that a level of about 16% CP was necessary for the optimal mobilization of fatty acids from body fat to provide substrates for milk fat synthesis in early lactation (Bines, 1982).

The higher milk protein yields found with the high protein treatments were consistent with the findings of Edwards et al (1980), but disagreed with the observation of Ha and Kennelly (1984) that higher levels of protein in the diet did not increase protein yields.

The significantly higher liveweights found on the high protein treatment coincided with the suggestion of Orskov et al (1977). He suggested that an increased protein supply increased the energy deficit of cows already in negative energy balance, since cows in negative energy balance required extra protein to match the available energy from the mobilized tissue, which was presumed to have a low protein content.

There were some indications that lame cows did not perform

as well as those who were not lame. For example, significant correlations were found between liveweight change and locomotion score and between condition score and locomotion score, when all 48 animals were included in the calculations, as shown in equations 3.4 and 3.5, respectively:

$r=-0.707$ ($p<0.05$)----------equation 3.4

$y=1.496-0.717(+0.227)x$

where y=overall liveweight change and

x=overall locomotion score

$r=-0.695$ ($p<0.05$)----------equation 3.5

$y=3.973-1.015(+0.332)x$

where y=overall condition score and

x=overall locomotion score

The effects of locomotion score on liveweight change and condition score may have partly been due to cows with poorer locomotion scores spending significantly less time eating and significantly more time lying. A cow whose locomotion score increased by one unit spent on average 41 minutes less time feeding (see Table 5.15). However, although negative correlations between locomotion score and feed were found these were not significant. There were no significant correlations between locomotion score and milk yield reponse (overall yield/14 day yield). Overall levels of lameness were relatively low, so the effects of treatment on lameness would have been unlikely to be manifested in terms of milk production. Additionally, as the diets were group fed any reduction in intake by the lame cows may have led to an increased intake by non-lame cows through the increased availability of feed.

Blood analyses

CK levels and serum GOT levels are indicators of muscle damage and tissue damage, respectively. Although significantly higher CK and serum GOT levels were found on the high protein treatments where locomotion scores were also significantly higher, no significant correlations between locomotion score and CK and serum GOT levels were found. Tissue damage associated with lameness would have been transient in some of the clinical cases of lameness, and as an average of the levels of each parameter found at the beginning and end of each period was used in the statistical analysis, a significant correlation

between the variables would not necessarily have been expected. The higher levels observed on the high protein treatments may have been due to the high protein diet either causing tissue damage in some part of the cow other than her feet, or resulting in high levels of serum GOT and CK through some other mechanism.

The mean Ca, Mg and P levels fell in the normal range as suggested by Topps and Thompson (1984). It is difficult to explain the higher levels of P found on the high protein treatments; possibly the high protein levels may have indirectly affected P and, or Ca metabolism through some intermediary factor, such as enzyme function. P and Ca intakes were the same on all treatments.

Hoof hardness in the toe sole region was positively correlated with Ca level in the blood. However, which variable is dependent and which is independent is unknown. Whether high Ca levels cause the hoof to become harder by some mechanism, or if they are indirectly related through some third variable is also unknown. The association requires further examination.

Thus in this trial there were problems in equating the blood parameters with lameness, and these blood measures did not appear to disclose any further information about a possible etiological factor associated with lameness. In a previous trial blood samples were taken from normal cows, cows with acute laminitis and cows with chronic laminitis, and various haematological and biochemical parameters, which included Ca, Mg, P and serum GOT levels, were compared (Maclean, 1970). With the exception of the serum GOT levels, which were significantly raised in the cows with chronic laminitis, no significant differences between the laminitic and normal cows were found. These findings seem to preclude the use of the blood parameters used in this trial as measures and diagnostic tools for lameness.

SUMMARY

The effects of protein level in a complete diet and Dutch hoof trimming on lameness were measured during weeks 3 to 26 on 48 autumn- calving cows. The four treatments were LT (161 g/kg crude protein, trimmed), LUT (161 g/kg, untrimmed), HT (198 g/kg, trimmed), HUT (198 g/kg, untrimmed). The protein levels were adjusted by substituting sugar beet for soya. ME intakes were maintained at the same level for all treatments by adjusting the intake of the high protein diet to that of the low protein, which was fed ad libitum. Cows were scored for their locomotion weekly on a scale of 1 to 5 (high scores indicated poorer locomotion), and hoof growth, wear and hardness measurements were recorded every eight weeks on the right hind outer claw. Hoof shape measurements were also recorded on the outer and inner claws of the right hind outer foot every eight weeks. Blood samples were taken at the beginning and end of each period and analysed for Ca, Mg, P, CK and serum GOT levels.

Trimming reduced and the high protein level significantly increased the locomotion score. Higher incidences of lameness were found both in the high protein treatments and in the untrimmed treatments; solar ulcers constituted 0.6 of all clinical cases.

Trimming also significantly increased hoof growth and reduced heel bulb hardness. Locomotion score was negatively correlated with mid sole hardness and with toe angle, and positively correlated with toe length.

No significant correlations between locomotion score and the blood parameters were found.

There were indications that lame cows did not perform as well as those who were not lame. Significant, negative correlations were found between livewight change and locomotion score, and between locomotion score and condition score.

CHAPTER 4

EXPERIMENT 3. The effect of concentrate to silage ratio and Dutch hoof trimming on lameness, hoof growth and wear, hoof shape and hoof hardness.

INTRODUCTION

In the first experiment it was suggested that the higher concentrate level increased locomotion score possibly through its higher protein intake (see Experiment 2), and possibly through its higher concentrate to silage ratio predisposing to laminitis. Similarly, Livesey and Fleming (1984) found that a low fibre, high starch diet led to a greater incidence of laminitis and solar ulcers than a high fibre diet. Acidosis has been implicated in the development of such laminitic/solar ulcer type syndromes. (Weaver, 1979; Andersson and Bergman, 1980).

Laminitis is thought to lead to the formation of poor quality horn (Edwards, 1982), and therefore it might be expected that hoof wear would be increased and hoof hardness decreased by the feeding of a high concentrate to silage ratio. However, evidence for this is scarce.

In Experiment 2 trimming was found to significantly increase net hoof growth. As one of the functions of trimming is to reduce toe length in order to help correct the weight distribution within and between the claws, such an effect of trimming on growth may reduce its potential for reducing lameness. This aspect requires further investigation. This experiment examined the effects of concentrate to silage ratio and Dutch hoof trimming on locomotion, clinical lameness and hoof growth and wear, shape and hardness. The relationships between the hoof measurements with lameness were also investigated.

MATERIALS AND METHODS

Treatments

Forty-eight cows were allocated in quartets according to projected calving date and parity (first lactation, second lactation and third lactation plus). Two cows from each quartet were chosen at random and had their feet trimmed to Dutch standards three weeks before the trial started. The Dutch trimming method is described in Appendix 2. Quartets of cows containing two trimmed and two untrimmed animals were balanced for the 14 day values of milk yield and liveweight, which are shown in Appendix 10. Each of the two trimmed cows were allocated at random to one of the two dietary treatments, and the untrimmed cows were similarly allocated. The cattle started on trial on average 15 days after calving (range 9 to 29 days). The four treatments were:

38:62 concentrate to silage ratio in the total dry matter, hooves trimmed (LCT)

38:62 concentrate to silage ratio in the total dry matter, hooves untrimmed (LCUT)

63:37 concentrate to silage ratio in the total dry matter, hooves trimmed (HCT)

63:37 concentrate to silage ratio in the total dry matter, hooves untrimmed (HCUT).

On average over the trial period the low and the high concentrate ratio diets contained 11.2 MJ/kg DM and 169 g/kg CP, and 11.9 MJ/kg DM and 180 g/kg CP in the total dry matter, repectively. All cattle received 1 kg concentrate at the am and pm milking. The remainder of the diet was fed as a complete diet, and the silage and the low and high ratio mixes were mixed using a paddle type West mixer wagon. The concentrate to silage ratios were adjusted by substituting the mixes with silage. Mean ME intakes and mean CP intakes were maintained at the same level in both groups by adjusting twice weekly the high concentrate ratio diet ME intake to that of the low concentrate ratio ME intake, which was fed ad libitum. Since the energy density of the high concentrate diet was greater than that of the low concentrate diet, less total DM per unit of ME intake was

required for the high concentrate diet. Therefore, the CP concentration of the high concentrate diet was higher than that of the low concentrate diet, so that both dietary treatments received the same CP intake per unit of ME intake.

Livestock and management

Forty-eight British Friesan cows were used in a continuous design experiment lasting from weeks 3 to 26 of lactation. The calving dates ranged from 21 August to 23 October. The trial lasted from 19 September 1985 to 17 April 1986.

After calving during the pre-trial period, the cattle were housed in a cubicle building and offered grass silage ad libitum in a feed passage and 6 kg concentrates/day in the parlour.

During the trial period the cattle were housed in a cubicle building, the cubicles being bedded with sawdust weekly. The concrete passages were scraped twice daily. The cows walked through a 5% formalin footbath weekly.

The silage was made from first cut perennial ryegrass, cut on 25 May 1985 with a drum mower, wilted for 24 hours and harvested with a precision chop forage harvester. Formic acid (Add-F, BP International Ltd., 850g formic acid/l) was applied at 2.3 litres/tonne, and the silage was ensiled in an unroofed bunker and sheeted with black polythene.

The chemical compositions of the silage, low and high concentrate mixes and parlour concentrate, and of the total low concentrate to silage ratio and high concentrate to silage ratio diets are shown in Tables 4.10 and 4.11. The physical ingredients of the parlour concentrate and low and high concentrate mixes are shown in Table 4.12.

Methods and records

Cow performance

The ingredients of the low and high concentrate mixes (barley, soya, sugar beet and minerals and vitamins) were sampled

Table 4.10 Chemical compositions of silage, mixes* and parlour concentrate (g/kg DM)

	silage	low conc@ mix	high conc mix	parlour concentrate
Oven dry matter (g/kg)	190	847	824	858
Crude protein	135	242	205	205
Organic matter	915			
DOMD (in vitro)	662	754	786	741
Predicted ME (MJ/kg DM)	10.5	12.1	12.6	13.3
Ammonia N (g/kg DM)	106			
pH	4.1			
Calcium	5.4	20.5	10.0	10.4
Phosphor us	3.0	8.7	6.5	7.8
Magnesium	1.9	7.2	4.2	5.8

@ conc=concentrate

Table 4.11 Chemical compositions of total diets (g/kg DM)

	low concentrate diet	high concentrate diet
Oven dry matter (g/kg)	422	606
Crude protein	169	180
DOMD (in vitro)	693	738
Predicted ME (MJ/kg DM)	11.2	11.9
Calcium	9.7	8.4
Phosphor us	5.4	4.9
Magnesium	3.5	3.6

Table 4.12 Physical Ingredients of Concentrate (kg/1000 kg)

Barley	250
Maize gluten	200
Wheat	200
Wheat feed	80
Soya	150
Fishmeal	20
Molasses	50
Fat supplement	20
Dicalcium phosphate	5
Minerals and vitamins	25

Physical ingredients of low and high ratio mixes (g/1000 kg)

	Low ratio mix	High ratio mix
Barley	614	614
Soya	39	234
Sugar beet	312	117
Minerals and vitamins	35	35

for dry matter weekly and the silage daily. Weekly samples of the silage, fortnightly samples of the parlour concentrate and monthly samples of the concentrate mix ingredients were taken for chemical analysis. The techniques for these analyses were those of Alexander and McGowan (1966; 1969), (see Appendix 5).

The recording of milk yields and compositions, liveweights and condition scores were carried out as outlined in Chapter 2. In addition to lameness, records of fertility, mastitis and other aspects of health were kept.

Locomotion scores, hoof shape, hoof growth and wear, and hoof hardness measurements were recorded as described in Chapters 2 and 3.

Statistical analysis

The results were analysed as a 2x2 factorial design (2 trimming and 2 concentrate to silage treatments) using the Edex statistical package (Hunter, Patterson and Talbot, 1973, Edinburgh). Data for the cows were analysed in three, eight week periods. Three missing values were used in the analyses of the milk yields and milk compostitions, since one cow from each of the treatments LCT, LCUT and HCUT were mistakenly dried off too late giving a dry period of less than two weeks. This led to abnormally low milk yields in these cows. Minitab (Pennsylavania State University, 1980) was used to calculate correlation and regression coefficients between various measurements. As in the previous trial an analysis of variance was carried out to investigate whether it was necessary to adjust a current incident of lameness for a particular cow, for a previous incident of lameness in the Chi-squared test, see Chapter 2. It was found that using the covariate made no significant improvement to the analysis.

RESULTS

Data are presented as four, five week periods commencing at 3 weeks post calving.

Locomotion score and clinical incidence of lameness

Trimming reduced locomotion scores in all three periods, although not significantly. The cows on the high concentrate diet

had higher locomotion scores than those on the low concentrate diet, and significant differences were found in periods 2 and 3 and for the overall means. No interactions between concentrate:silage ratio and trimming were found (see Table 4.13). There was an increasing divergence between the low and high concentrate diets over the trial period, whereas there were some indications that the differences in locomotion score between the trimmed and untrimmed treatments decreased as the trial progressed.

The number of clinical cases of lameness per cow week and the diagnoses of the clinical cases are shown in Tables 4.14 and 4.15, respectively. Of the 77 cases of clinical lameness, 32 were diagnosed as being of a solar nature and 29 were diagnosed as foot rot. In 14 examinations cases of both foot rot and solar problems were found to be present simultaneously (ie. 28 cases in total). When two types of lesions were observed in the same claw at the same examination, 2 cases of clinical lameness were recorded. The clinical incidence of lameness was significantly ($p<0.001$) associated with treatment type, see Table 4.14.

Correlation coefficients and regression equations for locomotion score on various hoof measurements are shown in Table 4.16.

Hoof growth and hoof wear

Hoof growth and wear means for periods 1 to 3 and overall are shown in Table 4.17.

Trimming significantly increased hoof growth and net hoof growth in periods 2 and 3 ($p<0.05$) and overall ($p<0.01$). Trimming reduced hoof wear, although not significantly.

No significant effects of concentrate to silage ratio on hoof growth or hoof wear were found, but there was a tendency of increasing hoof wear on the high concentrate diet over the trial period.

Hoof wear was greater than hoof growth for treatment LCUT in

Table 4.13 Locomotion score means

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 1	1.57	1.76	1.71	1.93	0.13NS	0.13NS	0.18NS
Period 2	1.59a@	1.67ab	1.85ab	2.04b	0.14*	0.14NS	0.20NS
Period 3	1.53a	1.61a	1.96b	2.10b	0.12*	0.12NS	0.17NS
Means of periods 1 to 3	1.56a	1.68a	1.84ab	2.02b	0.12*	0.12NS	0.16 NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 4.14 Incidence of lameness

	LCT	LCUT	HCT	HCUT
Number of clinical cases per cow week@	0.028	0.069	0.056	0.115

main treatment effects *** (Chi-squared analysis)

@ cows scoring 3 or more divided by the number of cow weeks (12x24)

Table 4.15 Diagnoses of clinical cases of lameness@

	Treatments			
	LCT	LCUT	HCT	HCUT
Number of examinations	8	20	16	33
Solar bruising	0	1	0	4
Solar ulcer	1	3	7	4
Double sole/sole separation	0	8	0	3
Solar penetration	0	1	0	0
Interdigital growth	2	0	0	8
Foot rot@	3	7	8	11
Leg injury	2	0	1	3

@ cows with locomotion scores of 3 or more. Where a cow has two types of lesion present simultaneously, both lesions are recorded in the table

@ foot rot=dermatitis interdigitalis with underrunning

Table 4.16 Significant correlation coefficients and regression equations for locomotion score (Y) on hoof shape measurements (X).

Locomotion score (Y) on outer toe length (X) @	r=+0.390 (p<0.01)	y=0.15+0.18(+0.06)X
Locomotion score (Y) on inner toe length (X) @	r=+0.334 (p<0.05)	y=-0.62+0.28(+0.12)X
Locomotion score (Y) on outer toe angle (X) @	r=-0.331 (p<0.05)	y= 2.16-0.01(+0.01)X
Locomotion score (Y) on inner toe angle (X) @	r=-0.420 (p<0.01)	y= 2.90-0.03(+0.01)X
Locomotion score (Y) on outer heel height (X) period 3	r=-0.279 (p<0.05)	y= 2.49-0.23(+0.12)X
Locomotion score (Y) on outer heel length (X) @	r=-0.345 (p<0.05)	y= 3.09-0.38(+0.15)X
Locomotion score (Y) on inner heel length (X) @	r=-0.283 (p<0.05)	y= 2.89-0.33(+0.17)X
Locomotion score (Y) on differences in claw length (X) period 3	r=+0.279 (p<0.05)	y= 1.66+0.20(+0.11)X

@ values calculated using the means of periods 1 to 3

Table 4.17 Hoof growth and hoof wear means

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 1							
Hoof growth (cm/month)	0.55	0.46	0.49	0.42	0.09NS	0.09NS	0.13NS
Hoof wear (cm/month)	0.44	0.47	0.30	0.36	0.10NS	0.10NS	0.14NS
Net hoof growth (cm/month)	0.11	-0.01	0.19	0.06	0.11NS	0.11NS	0.16NS
Period 2							
Hoof growth (cm/month)	0.56a@	0.44ab	0.54ab	0.41b	0.05NS	0.05*	0.07NS
Hoof wear (cm/month)	0.44	0.44	0.36	0.50	0.09NS	0.09NS	0.13NS
Net hoof growth (cm/month)	0.12ab	0.00ab	0.19a	-0.08b	0.09NS	0.09*	0.13NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 4.17 Hoof growth and hoof wear means

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 3							
Hoof growth (cm/month)	0.69a@	0.53ab	0.59ab	0.46b	0.05NS	0.05*	0.07NS
Hoof wear (cm/month)	0.41	0.51	0.47	0.56	0.06NS	0.06NS	0.09NS
Net hoof growth (cm/month)	0.28a	0.03b	0.12ab	-0.10b	0.08NS	0.08**	0.12NS
Means of periods 1 to 3							
Hoof growth (cm/month)	0.60a	0.48bc	0.54ab	0.43c	0.03NS	0.03**	0.05NS
Hoof wear (cm/month)	0.43	0.47	0.38	0.47	0.04NS	0.04NS	0.06NS
Net hoof growth (cm/month)	0.18a	0.01b	0.16a	-0.04b	0.05NS	0.05**	0.08NS

@ means with different subscripts are significantly different ($p<0.05$)

period 1 and for treatment HCUT in periods 2 and 3 and overall. For other treatments and other periods hoof growth exceeded hoof wear.

Hoof shape

Hoof shape means for periods 1 to 3 and overall are shown in Table 4.18. The initial and final toe length and heel length means are shown in Appendix 11.

Trimming significantly decreased outer claw toe length in periods 1 and 2 and overall and increased outer toe angle in all periods. Inner toe lengths were significantly lower on the trimmed treatments in period 1 and inner toe angles were significantly steeper in all periods and overall. There were no signficant effects of concentrate to silage ratio on toe length of toe angle.

Neither trimming nor the concentrate to silage ratio significantly affected outer claw heel height or outer and inner claw heel lengths.

Trends of increasing outer claw toe length and decreasing outer claw toe angle over the trial period were found in the trimmed treatments. Increases in inner toe length over the 24 week period were found in all treatments, as were decreases in outer claw heel height. With the exception of treatment HCT, there was a trend of decreasing outer claw heel length.

The correlation coefficients and regression equations for locomotion score and various hoof shape measurements, and for angle of toe and length of toe are shown in Tables 4.16 and 4.19, respectively.

Hoof hardness

Hoof hardness means are shown in Table 4.20. The initial and final hoof hardness means are shown in Appendix 12.

The abaxial wall toe region in all periods and overall, and

Table 4.18 Hoof shape means

	Treatments				SED		
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 1							
Length of toe (outer) (cm)	8.29a@	9.41b	8.48a	9.73b	0.18NS	0.18***	0.25NS
Length of toe (inner) (cm)	8.15a	8.43ab	8.23ab	8.63b	0.17NS	0.17*	0.23NS
Angle of toe (outer) (o)	41.2a	25.5b	39.3a	29.7b	2.87NS	2. 87***	4.06NS
Angle of toe (inner) (o)	43.4ab	39.8ab	44.6a	38.4b	1.81NS	1.18*	2.57NS
Height of heel (outer) (cm)	3.48	3.18	3.38	3.26	0.12NS	0.12NS	0.16NS
Length of heel (outer) (cm)	3.68	3.43	3.56	3.65	0.12NS	0.12NS	0.18NS
Length of heel (inner) (cm)	3.57	3.24	3.44	3.46	0.10NS	0.10NS	0.17NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 4.18 Hoof shape means

(continued)

	Treatments				SED		
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 2							
Length of toe (outer) (cm)	8.54a@	9.45bc	8.83ac	9.75b	0.26NS	0.26**	0.40NS
Length of toe (inner) (cm)	8.38	8.61	8.42	8.82	0.16NS	0.16NS	0.23NS
Angle of toe (outer) (o)	38.7a	23.4b	37.4a	29.2b	2.62NS	2.62***	3.70NS
Angle of toe (outer) (cm)	41.7ab	37.8b	44.0a	36.3b	1.96NS	1.96**	2.77NS
Height of heel (outer) (cm)	3.36	3.07	3.26	3.24	0.09NS	0.09NS	0.15NS
Length of heel (outer) (cm)	3.62	3.38	3.48	3.50	0.13NS	0.13NS	0.18NS
Length of heel (inner) (cm)	3.44	3.17	3.40	3.37	0.10NS	0.10NS	0.15NS

@ means with different subscripts are significantly different

Table 4.18 Hoof shape means

(continued)

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 3							
Length of toe (outer) (cm)	8.93	9.53	9.07	9.59	0.31NS	0.31NS	0.44NS
Length of toe (inner) (cm)	8.54	8.78	8.57	8.91	0.16NS	0.16NS	0.22NS
Angle of toe (outer) (o)	36.8a@	22.8b	36.8a	28.6ab	3.13NS	3.13**	4.42NS
Angle of toe (inner) (o)	42.0ab	37.6a	45.1b	37.2a	1.91NS	1.91**	2.70NS
Height of heel (outer) (cm)	3.12	2.68	3.12	3.18	0.20NS	0.20NS	0.29NS
Length of heel (outer) (cm)	3.43	2.91	3.62	3.4	0.23NS	0.23NS	0.35NS
Length of heel (inner) (cm)	3.30	3.25	3.48	3.23	0.14NS	0.14NS	0.20NS

@ means with different subscripts are significantly different

Table 4.18 Hoof shape means

(continued)

	Treatments				SED		
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Means of periods 1 to 3							
Length of toe (outer) (cm)	8.59a@	9.46bc	8.79ac	9.69b	0.24NS	0.24***	0.34NS
Length of toe (inner) (cm)	8.36	8.61	8.41	8.79	0.17NS	0.17NS	.4NS
Angle of toe (outer) (o)	38.9a	23.9b	37.8a	29.1b	2.65NS	2.65***	3.74NS
Angle of toe (inner) (o)	42.4ac	38.4bc	44.6a	37.3b	1.69NS	1.69**	2.39NS
Height of heel (outer) (cm)	3.31	2.99	3.25	3.23	0.10NS	0.10NS	0.16NS
Length of heel (outer) (cm)	3.58	3.24	3.56	3.54	0.12NS	0.12NS	0.17NS
Length of heel (inner) (cm)	3.44	3.22	3.44	3.35	0.10NS	0.10NS	0.14NS

@ means with different subscripts are significantly different

Table 4.19 Significant correlation coefficients and regression equations for angle of toe (Y) on length of toe (X)

	Outer claw	Inner claw
Angle of toe (Y) on length of toe (X) period 1	r=-0.702 (p<0.001) y=116.4-9.2(+1.39)X	r=-0.418 (p<0.01) y=82.7-4.9(+1.59)X
Angle of toe (Y) on length of toe (X) period 2	r=-0.728 (p<0.001) y=105.9-8.1(+1.12)X	r=-0.542 (p<0.001) y=100.9-7.1(+1.63)X
Angle of toe (Y) on length of toe (X) period 3	r=0.683 (p<0.001) y=109.9-8.5(+1.34)X	r=-0.741 (p<0.01) y=129.1-10.2(+1.36)X
Angle of toe (Y) on length of toe (X) mean of periods 1 to 3	r=-0.747 (p<0.001) y=116.4-9.1(+1.21)X	r=-0.613 (p<0.001) y=108.1-7.9(+1.50)X

Table 4.20 Hoof hardness means (Shore A units, scale 0 to 100)

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 1							
Abaxial wall toe	88.7a@	88.6ab	86.3ab	85.7b	1.04*	1.04NS	1.47NS
Abaxial wall mid	86.8	87.5	86.0	84.3	1.12NS	1.12NS	1.59NS
Sole toe	86.3a	80.5b	82.9ab	81.3b	1.34NS	1.34**	1.90NS
Sole mid	77.3a	76.4ab	71.8b	72.3ab	1.92*	1.92NS	2.72NS
Heel bulb centre	40.7	42.0	41.9	43.8	1.47NS	1.47NS	2.07NS
Period 2							
Abaxial wall toe	90.7a	91.9a	86.9b	86.2b	1.17***	1.17NS	1.66NS
Abaxial wall mid	89.7ac	90.4a	87.3bc	85.6b	1.06**	1.06NS	1.49NS
Sole toe	87.0a	86.2ab	84.3ab	83.3b	1.03*	1.03NS	1.45NS
Sole mid	78.8ab	80.3a	75.0b	75.4ab	1.83*	1.83NS	2.58NS
Heel bulb centre	38.7	39.1	40.2	40.3	1.75NS	1.75NS	2.47NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 4.20 Hoof hardness means (Shore A units, scale 0 to 100)

(continued)

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 3							
Abaxial wall toe	87.7ab@	88.4a	84.5b	85.5ab	1.16*	1.16NS	1.64NS
Abaxial wall mid	86.2	87.1	85.3	86.3	1.01NS	1.01NS	1.43NS
Sole toe	84.7ab	87.3a	81.9b	81.7b	1.32**	1.32NS	1.86NS
Sole mid	79.2ab	81.4a	75.3b	76.6ab	1.70*	1.70NS	2.41NS
Heel bulb centre	36.7	38.2	37.0	33.9	2.71NS	2.71NS	3.83NS
Means of periods 1 to 3							
Abaxial wall toe	89.0a	89.6a	85.9b	85.8b	0.90***	0.90NS	1.27NS
Abaxial wall mid	87.6ab	88.3a	86.2ab	85.4b	0.79*	0.79NS	1.11NS
Sole toe	86.0a	84.7ab	83.0b	82.1b	0.97**	0.97NS	1.37NS
Sole mid	78.4ac	79.2a	74.2b	74.7bc	1.47**	1.47NS	2.07NS
Heel bulb centre	38.7	39.8	39.7	39.3	1.53NS	1.53NS	2.17NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 4.21 Significant correlation coefficients and regression equations for locomotion score (Y) on hoof hardness measurements (X).

Locomotion score (Y) on abaxial wall toe hardness (X) @	r=-0.285 ($p<0.05$)	$y=4.41-0.03(\pm0.02)X$
Locomotion score (Y) on abaxial wall mid hardness (X) period 2	r=-0.281 ($p<0.05$)	$y=3.52-0.02(\pm0.01)X$
Locomotion score (Y) on sole toe (X) period 3	r=-0.356 ($p<0.05$)	$y=4.61-0.03(\pm0.01)X$
Locomotion score (Y) on sole mid (X) period 2	r=-0.280 ($p<0.05$)	$y=3.16-0.02(\pm0.01)X$

@ values calculated from means of periods 1 to 3

the abaxial wall mid region in period 2 and overall were significantly harder on the low concentrate:silage ration compared to on the high concentrate to silage ratio. The sole toe region in periods 2 and 3 and the sole mid region in all periods were also significantly harder on the low concentrate treatments.

Trimming significantly increased the hardness of the sole toe region in Period 1. No other effects of trimming were found.

The overall abaxial wall toe region was harder than the overall abaxial wall mid, except on treatment HCT. The abaxial wall mid was in turn harder than the sole toe, which was harder than the sole mid region. The heel bulb centre was the softest region.

There was a trend of decreasing hardness of the heel bulb centre over the trial period, which was particularly marked in the high concentrate group.

The correlation coefficients and regression equations for locomotion score and various hoof hardness measurements are shown in Table 4.21.

Cow performance

The mean daily intakes of dry matter, and the calculated ME and CP intakes are shown in Table 4.22.

As in the previous trials, with the exception of the liveweight change and condition score change analyses, the analyses of the production parameters were adjusted using covariates based on the 14 day post-partum values.

The results of the milk production parameters are shown in Table 4.23. The milk yields of the cows on the high concentrate to silage ratio were significantly ($p<0.01$) higher as compared to those on the low concentrate to silage ratio in periods 1, and 2 and overall. A significant ($p<0.05$) interaction between the concentrate to silage ratio and trimming was also found in period 1. There were significant ($p<0.05$) interactions between

Table 4.22 Mean daily intakes of dry matter and the calculated ME and CP intakes

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 1							
Complete diet DM intake (kg/day)	15.1	15.2	14.2	14.2	0.55NS	0.55NS	0.77NS
Concentrate DM intake (kg/day)	1.7	1.7	1.7	1.7			
Total DM intake (kg/day)	16.8	16.9	15.9	15.9	0.55NS	0.55NS	0.77NS
Total ME intake (MJ/day)	189	190	191	187	5.01NS	5.01NS	7.16NS
Total CP intake (g/day)	2789	2860	2880	2917	62.04NS	62.04NS	85.56NS
Conc:silage ratio	36:64		64:36				

Table 4.22 Mean daily intakes of dry matter and the calculated

(continued) ME and CP intakes

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 2							
Complete diet DM intake (kg/day)	13.2	13.3	12.2	12.3	0.51NS	0.51NS	0.72NS
Concentrate DM intake (kg/day)	1.7	1.7	1.7	1.7			
Total DM intake (kg/day)	14.9	15.0	13.9	14.0	0.51NS	0.51NS	0.72NS
Total ME intake (MJ/day)	167	170	164	171	5.09NS	5.09NS	7.86NS
Total CP intake (g/day)	2550	2560	2529	2589	67.27NS	67.27NS	95.13NS
Conc:silage ratio	39:61		63:37				

Table 4.22 Mean daily intakes of dry matter and the calculated ME and CP intakes (continued)

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Period 3							
Complete diet DM intake (kg/day)	12.4	12.6	11.7	12.0	0.45NS	0.45NS	0.64NS
Concentrate DM intake (kg/day)	1.7	1.7	1.7	1.7			
Total DM intake (kg/day)	14.1	14.3	13.4	13.7	0.45NS	0.45NS	0.64NS
Total ME intake (MJ/day)	158	161	156	163	5.69NS	5.69NS	8.04NS
Total CP intake (g/day)	2385	2470	2432	2528	62.04NS	62.04NS	85.56NS
Conc:silage ratio	38:62		62:38				

Table 4.22 Mean daily intakes of dry matter and the calculated ME and CP intakes (continued)

	Treatments					SED	
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Means of periods 1 to 3							
Complete diet DM intake (kg/day)	13.6	13.7	12.7	12.8	0.30NS	0.30NS	0.48NS
Concentrate DM intake (kg/day)	1.7	1.7	1.7	1.7			
Total DM intake (kg/day)	15.3	15.4	14.4	14.5	0.30NS	0.30NS	0.48NS
Total ME intake (MJ/day)	171	174	170	174	4.03NS	4.03NS	5.56NS
Total CP intake (g/day)	2575	2630	2614	2678	59.80NS	59.80NS	64.7NS
Conc:silage ratio	38:62		63:37				

Table 4.23 Milk yield and milk compos ition means

	Treatments				SED		
	LCT	LCUT	HCT	HCUT	C:S ratio(R)	trim (T)	RxT
Period 1							
Milk yield (kg/day)	24.4a@	23.9a	25.2a	27.7b	0.70**	0.70NS	0.98*
Milk fat content (g/kg)	40.8ab	43.0a	42.8ab	40.5b	0.86NS	0.86NS	1.22*
Milk fat yield (g/day)	986a	1027ac	1083bc	1113b	29.8**	29.8NS	42.2NS
Milk protein content (g/kg)	30.9b	31.7ab	33.1a	31.8ab	0.55*	0.55NS	0.77NS
Milk protein yield (g/day)	763a	757a	819b	883c	18.7***	18.7NS	26.5NS
Period 2							
Milk yield (kg/day)	20.6a	20.7a	21.0a	23.5b	0.81**	0.81NS	1.15NS
Milk fat content (g/kg)	38.6a	41.0ab	42.3b	40.2ab	1.02NS	1.02NS	1.44*
Milk fat yield (g/day)	798a	838a	879ab	949b	31.1**	31.1NS	44.0NS
Milk protein content (g/kg)	31.4a	32.7ab	34.3ab	33.6b	0.75*	0.75NS	1.06NS
Milk protein yield (g/day)	657a	675a	706a	786b	23.2**	23.2*	32.8NS

@ means with different subscripts are significantly different ($p<0.05$)

Table 4.23 Milk yield and milk compos ition means

(continued)

	Treatments				SED		
	LCT	LCUT	HCT	HCUT	C:S ratio(R)	trim(T)	RxT
Period 3							
Milk yield (kg/day)	17.4	17.3	17.4	19.2	0.95NS	0.95NS	1.35NS
Milk fat content (g/kg)	41.6	41.5	43.0	40.2	1.21NS	1.21NS	1.71NS
Milk fat yield (g/day)	732	708	732	780	36.2NS	36.2NS	51.2NS
Milk protein content (g/kg)	32.8	33.6	34.8	34.4	0.71NS	0.71NS	1.00NS
Milk protein yield (g/day)	580	584	591	660	28.0NS	28.0NS	39.6NS
Means of periods 1 to 3							
Milk yield (kg/day)	20.8a	20.6a	21.2a	23.5b	0.78**	0.78NS	1.10NS
Milk fat content (g/kg)	40.3	41.8	42.7	40.3	0.94NS	0.94NS	1.32NS
Milk fat yield (g/day)	839a@	858a	898ab	947b	29.4**	29.4NS	41.5NS
Milk protein content (g/kg)	31.7a	32.7ab	34.1b	33.2ab	0.65*	0.65NS	0.91NS
Milk protein yield (g/day)	667a	672a	705a	777b	21.4**	21.4NS	30.3NS
Milk lactose content (g/kg)	47.2a	46.5b	47.5a	47.7a	0.24**	0.24NS	0.33NS
Milk S-N-F content (g/kg)	86.6a	86.7ac	88.9b	88.5bc	0.62**	0.62NS	0.86NS
Milk TS content (g/kg)	126.9a	128.5ab	131.6b	128.7ab	1.29*	1.29NS	1.82NS

@ means with different subscripts are significantly different ($p<0.05$)

concentrate to silage ratio and trimming on the milk fat contents in periods 1 and 2. Milk fat yields were significantly higher on the high concentrate to silage ratio treatments in periods 1 and 2 and overall. The high concentrate to silage ratio significantly ($p<0.05$) increased the milk protein contents and milk protein yields in periods 1 and 2 and overall, and the milk protein yields were also significantly higher on the untrimmed treatments in period 2. Both the overall milk lactose contents and the overall milk solids-not-fat contents were significantly ($p<0.01$) increased by the high concentrate to silage ratio treatment. Significantly ($p<0.05$) higher milk total solids contents were found on the high concentrate to silage ratio treatments.

There were no significant effects of trimming or concentrate to silage ratio on liveweight or liveweight change. In period 2 the condition scores of the cows on the trimmed treatments were significantly ($p<0.05$) higher as compared to those of the cows on the untrimmed treatments. No further treatment effects on condition score or condition score change were found (see Tables 4.24 and 4.25).

The correlation coefficients and regression equations for various production parameters on locomotion score are presented in Table 4.26. With the exception of the significant correlation between condition score and locomotion score, when all 48 animals were used in the calculation, significant correlations were only found within treatments. Significant correlations between milk production parameters and locomotion score were found only for the two untrimmed treatments.

Table 4.24 Liveweight and liveweight change means

	Treatments				SED		
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim (T)	RxT
Period 1							
Liveweight (kg)	569	564	568	559	5.35NS	5.35NS	7.56NS
Liveweight change (kg/day)	+0.17	+0.03	+0.13	-0.02	0.14NS	0.14NS	0.19NS
Period 2							
Liveweight (kg)	575	565	570	571	8.63NS	8.63NS	12.20NS
Liveweight change (kg/day)	+0.05	+0.12	+0.09	+0.19	0.11NS	0.11NS	0.15NS
Period 3							
Liveweight (kg)	581	572	575	578	10.54NS	10.54NS	14.90NS
Liveweight change (kg/day)	+0.13	+0.19	+0.23	+0.31	0.11NS	0.11NS	0.16NS
Means of periods 1 to 3							
Liveweight (kg)	575	567	571	569	7.86NS	7.86NS	11.11NS
Liveweight change (kg/day)	+0.12	+0.11	+0.15	+0.16	0.07NS	0.07NS	0.10NS

Table 4.25 Condition score and condition score change means

	Treatments				SED		
	LCT	LCUT	HCT	HCUT	C:S ratio(R)	trim(T)	RxT
Period 1							
Condition score	2.17	2.08	2.15	2.05	0.06NS	0.06NS	0.08NS
Condition score change	-0.005	-0.018	+0.005	-0.015	0.01NS	0.01NS	0.02NS
Period 2							
Condition score	2.18a@	1.99ab	2.07ab	1.87b	0.10NS	0.10*	0.13NS
Condition score change	-0.012	+0.009	+0.003	+0.001	0.01NS	0.01NS	0.02NS
Period 3							
Condition score	2.16	2.10	2.00	1.98	0.12NS	0.12NS	0.17NS
Condition score change	-0.002	+0.022	-0.044	+0.028	0.03NS	0.03NS	0.04NS
Means of periods 1 to 3							
Condition score	2.17	2.06	2.07	1.97	0.08NS	0.08NS	0.12NS
Condition score change	-0.006	+0.004	-0.012	+0.005	0.01NS	0.01NS	0.02NS

@ means with different subscripts are significantly different

Table 4.26 Significant correlation coefficients and regression equations for various production parameters (Y) on locomotion score (X)

Condition score (Y) on locomotion score (X) using all 48 cows $	r=-0.566 (p<0.001)	y=3.33-0.71(±0.15)X
Milk yield response (Y) on locomotion score (X) for LCUT@	r=-0.544 (p<0.05)	y=1.08-0.13(±0.07)X
Milk protein yield (Y) on locomotion score (X) for HCUT	r=-0.661 (p<0.05)	y=116.2-19.1(±7.23)X
Condition score (Y) on locomotion score (X) for LCT	r=-0.584 (p<0.05)	y=4.03-1.13(±0.50)X
Condition score (Y) on locomotion score (X) for LCUT	r=-0.803 (p<0.001)	y=3.70-0.86(±0.20)X
Condition score change (Y) on locomotion score (X) for LCUT	r=-0.584 (p<0.05)	y=0.48-0.23(±0.10)X
Liveweight change (Y) on locomotion score (X) for LCUT	r=-0.598 (p<0.001)	y=0.52-0.24(±0.10)X

$ values calculated using the means of periods 1 to 3 for all 48 cows.

@ milk yield response=mean yield/14 day yield; LCUT=low concentrate to silage ratio treatment; values calculated using the means of periods 1 to 3.

DISCUSSION

Locomotion scores and clinical incidence of lameness

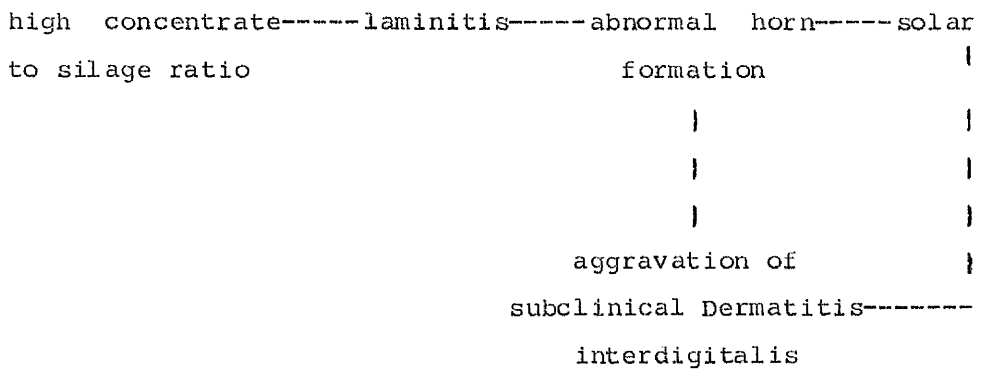
The higher locomotion scores found on treatments HCT and HCUT in this trial agree with the results from the first trial. The higher concentrate to silage ratio may have resulted in higher locomotion scores by predisposing to laminitis, which in turn would have led to increases in the frequency of solar problems, and may additionally have aggravated any Dermatitis interdigitalis that was already present in some of the cows. The greater number of clinical cases of lameness, due to solar problems, which were likely to have resulted from laminitis (solar bruising, solar ulcer and double sole/sole separation), agrees with the findings of Peterse et al (1984), who found that the incidence of solar ulcers was twice as high in a group of cows fed a diet with a concentrate to roughage ratio of 50:50, as compared with a group who received a diet mainly composed of roughage.

Laminitis probably predisposed to solar ulcers and other solar problems through its influence on hoof horn formation, as described in Chapter 3. Laminitis may also have aggravated Dermatitis interdigitalis which was present in many of the cows in both concentrate to silage ratio groups at the start of the trial period; pitting of the heel and the appearance of parallel grooves were observed in many of the cows when the initial measurements were made. The high incidence of Dermatitis interdigitalis was probably due to the exceptionally wet summer, which preceded the trial period; high rainfall and high atmospheric moisture levels have been associated with high incidences of Dermatitis interdigitalis (Smedegaard, 1963; Baggott and Russell, 1982).

Bacteroides nodosus, an important etiological factor in Dermatitis interdigitalis, spreads from the epidermis of the inter-digital skin to the heel bulbs of the adjacent claws, and where the germinal layer in the epidermis is affected, the connection between the corium and bulbar horn may become disrupted. Such heel horn erosion undermines the bulbar horn, resulting in the appearance of fissures running from the axial

region backwards to the abaxial region (Toussaint Raven, 1985), as was observed in many of the clinical cases in this trial. Erosion may cause underrunning of the heel and sole, and it has been suggested that underrunning is more likely to occur if solar keratinization is abnormal (Livesey and Fleming, 1984). Laminitis, which may have occurred as the result of feeding the high concentrate to silage ratio diets, would have led to the formation of such incompetent horn due to faulty keratinization. Underrunning of the heel and sole may then extend to the solar corium leading to ulceration. Positive correlations between heel erosion and haemorrhages at the "typical site" of solar ulcers have been found (Arkins et al.,1986). Bruising of the corium may also arise if the horny fissures press into the corium. Thus, this disease syndrome is progressive, with lameness becoming more severe in the latter stages of the disease, partly due to the development of solar problems and in particular solar ulcers. In this trial the locomotion scores of the high concentrate group increased as the trial progressed, whereas those of the low concentrate group decreased, and consequently the differences between the two groups in locomotion score became more significant over the 24 week period. Additionally, nine incidences of a combined solar ulcer/foot rot type of lameness were found in the high concentrate group, as compared to three cases in the low concentrate group. These results may indicate that on the low concentrate group Dermatitis interdigitalis remained at a low level because of the lack of aggravation by laminitis, whereas on the high concentrate group laminitis aggravated the Dermatitis interdigitalis so leading to a progressively more severe Dermatitis interdigitalis/underruning type of lameness, which was accompanied by the devlopment of solar ulcers. The progress of Dermatitis interdigitalis over the trial period is also reflected in the sharp decline in heel bulb hardness over the same period. The effect of the high concentrate:silage ratio on locomotion score and incidence of clinical lameness is summarised in the following figure:

Figure 4.1



Erosion of the heel bulb may also have reduced the ability of the heel bulb to act as an anti-concussive device, which would lead to the development of an abnormal gait, which would be then reflected in higher locomotion scores.

The improvement in locomotion with trimming, although not significant , was probably the consequence of a reduction in the outer and inner claw toe lengths, and of a better weight distribution between and within the claws. This was in accordance with the results of the previous trial. A significant reduction in the number of clinical cases with trimming would also have contributed to the lower locomotion scores on the trimmed treatments. It is likely that tissue bruising, caused by non-trimming would predispose to Dermatitis interdigitalis. Tissue bruising has been associated with a lowering of resistance to Phlegmona interdigitalis (Greenough, 1962), which is probably similar in etiology to Dermatitis interdigitalis. Trimming has been suggested as a preventative measure against Dermatitis interdigitalis (Smedegaard, 1964,b), and the prevention of severe lameness caused by ulceration, through the trimming of abnormal horn growth resulting from Dermatitis interdigitalis, has also been recommended (Toussaint Raven and Cor nelisse, 1971).

Hoof growth and hoof wear

The effects of trimming in increasing hoof growth agrees with the results of the previous trial (see Chapter 3).

Trimming may have reduced hoof wear by removing the older,

less elastic hoof tissue and exposing the more recently formed, more elastic tissue. It has been suggested that a more elastic hoof may be more resistant to the abrasive wear of concrete because of its ability to expand and contract (Clark and Rakes, 1982).

The increase in hoof wear of the cows on the high concentrate treatments over the trial period, was probably a reflection of the progressive nature of Dermatitis interdigitalis and of the effects of laminitis, which are similar in consequence. This is in accordance with the findings of the first trial, where it was suggested that laminitis caused by a higher concentrate to silage ratio might have led to lower levels of net growth by increasing wear rates. In the latter stages of Dermatitis interdigitalis, underrunning may cause local ulceration and extensive bruising of the corium, (Toussaint-Raven, 1971) and this in conjunction with laminitis leads to the production of malformed horn. Over a period of time depending on the current rates of hoof growth and wear, the malformed horn will reach the surface, and as the levels of malformed horn in the dorsal region build up (hoof wear was measured in the dorsal region of the abaxial wall) the susceptibility of the hoof to wear will similarly increase. It is uncertain as to which factors of hoof quality are important with respect to hoof wear. Although in this trial non significant correlations between dorsal hoof wear and abaxial wall toe and sole toe hardness were found, there were indications that in the heel region hoof wear was related to heel bulb hardness. Hoof elasticity and hoof compressive strength may also be related to hoof wear, and these factors require further investigation.

Hoof shape

The higher growth rates and lower wear rates found with the trimmed treatments were reflected in the gradual reduction in the differences between the trimmed and untrimmed treatments in outer claw toe length over the trial period. The initial effects of trimming on reducing outer claw toe length disppeared at 23 weeks (trimming was carried out 3 weeks before the trial started and the

toe length means for Period 3, shown in Table 4.18, are the means of the toe lengths recorded at 16 and 24 weeks after the start of the trial). This was reflected in the decrease over the trial period in the differences between the trimmed and untrimmed treatments in locomotion score; locomotion score and toe length were also significantly and positively correlated. Therefore, it appeared that trimming primarily affected locomotion score through its effects on toe length rather than through any of its additional effects on hoof shape and function. In the previous trial trimming appeared to influence locomotion score both through its effects on toe length and on other aspects of hoof shape and function. This discrepancy may have been due to any slight differences in trimming method employed by the two Dutch hoof trimmers.

With the exception of the correlation between angle of toe and length of toe of inner claw in period 1, very highly significant ($p<0.001$) correlations between angle of toe and length of toe were found, which indicated the suitability of length of toe as a predictor of angle of toe. In the previous trials the correlations were not so consistently significant and this may have been caused by less accuracy in the recording of toe lengths and toe angles.

The negative correlations between heel height and heel length with locomotion score were probably a reflection of the progressive and cumulative effects of Dermatitis interdigitalis on heel tissue structure over the trial period. These effects include erosion of the heel and the production of malformed heel horn which is likely to be highly susceptible to wear, and would therefore result in reduced heel heights and lengths. Trends of decreasing outer claw heel height and decreasing claw heel length (with the exception of treatment HCT) over the trial period were found.

Hoof hardness

The main dietary factor which has been associated with hoof hardness is the supply of sulphur amino acids and the contribution which they make to disulphide bonding in the keratin protein

(Hahn, 1979; Clark and Rakes, 1982). However, nutritional factors which predispose to laminitis may also be of consequence, for example, high concentrate to silage ratios have been associated with laminitis (Nilsson, 1963; Peterse, 1979; Weaver, 1979; Livesey and Fleming,1984). The action of endotoxins formed during acidosis leads to an insufficient supply of nutrients to the keratin forming cells, with subsequent faulty keratinization (Andersson and Bergman, 1980). This process has been associated with laminitis (Weaver, 1979). Therefore, the high concentrate to silage ratio by predisposing to laminitis and acidosis may have prevented adequate supplies of sulphur amino acids from reaching the hoof horn producing cells in the epidermis. This would then lead to lower levels of disulphide bonding in the keratin tissue, with softer horn therefore being formed in the solar and abaxial wall regions, as was found in this trial. (Protein intakes were maintained at the same level in all treatments). Although high protein levels were thought to predispose to laminitis in the previous trial, no effects of protein level on hoof hardness were found. However, there was evidence that locomotion, and therefore probably severity and duration of laminitis, was more significantly affected by concentrate to silage ratio than by protein level; with the exception of treatment LCUT, locomotion scores were higher in all periods in this trial as compared to the previous trial. Therefore, either the laminitis has to be of a severe or of a long-lasting nature, or its effects on hoof hardness have to be mediated through some other condition, for example, Dermatitis interdigitalis.

The decreases in both heel bulb centre hardness and outer claw heel shape measurements over the 24 weeks, together with the significant correlations between heel bulb centre hardness with heel height and heel length (see equation 4.1 and 4.2), reflect the effects of Dermatitis interdigitalis on heel tissue.

$r=+0.315$ ($p<0.05$)----------equation 4.1

$y=2.34+0.02(+0.01)x$

where y=outer claw heel height, period 3 and

x=outer claw heel bulb centre hardness, period 3

$r=+0.426$ ($p<0.01$)----------equation 4.2

$y=2.24+0.03(+0.01)x$

where y=outer claw heel length, mean of periods 1 to 3 and

x=outer claw heel bulb centre hardness, means of periods 1 to 3

Aggravation of sub-clinical Dermatitis interdigitalis by laminitis, causes the production of malformed horn, and is reflected in the horn being excessively soft, which predisposes to severe erosion and underrunning, which in turn leads to decreases in heel height and length. Since the effects of Dermatitis interdigitalis are progressive it would be expected that outer claw heel bulb centre hardness, height of outer heel and length of outer heel would decrease over the trial period. This was found with the exception of the length of heel on treatment HCT.

The negative correlation found between sole mid and sole toe hardness with locomotion score is in accordance with the negative correlation found between sole mid hardness and locomotion score in the previous trial, and similarly reflects the greater ability of a harder sole to protect the corium (locomotion score is likely to be indicative of the health of the corium). The negative correlations found between the abaxial wall measurements with locomotion score are probably due to softer horn being associated with a damaged corium, which is likely to result from laminitis and/or overburdening of the corium.

The significant effects of trimming on increasing the hardness of the sole toe region in period 1 could be attributed to the initial effect of trimming in shaping the axial solar region, which would allow the drainage of slurry away from the solar region. However, no further significant effects of trimming were found which could be related to the lack of any lasting effect of trimming on the shape of this region. This corresponds to the finding that trimming appeared to primarily influence locomotion through its reducing toe length, rather than to any additional effects on hoof shape and function. The lack of a consistent relationship between trimming and hardness may also have been accounted for by the low levels of slurry in the housing area due to twice daily scraping of the passageways.

Cow performance

In this trial mean ME intakes were maintained at the same level for both dietary treatments, whereas in previous trials, which have examined the effects of the concentrate to forage ratio on production, no adjustments were made to equalize ME intakes. This may have explained the discrepancy in the milk yield results of this trial with those of other trials. In this trial, higher milk yields were found on the high concentrate to silage ratio treatments, and this agrees with other trials where higher milk yields were associated with high concentrate diets (Sutton et al, 1980; Phipps et al, 1984).

Increases in the proportion of concentrates in the diet have been related to depressions in milk fat content (Sutton et al, 1980; DePeters and Smith, 1984). These depressions have been associated with an increase in the total acid concentration in the rumen and a decrease in pH, which is thought to favour a switch from acetate to propionate production with subsequent stimulation of insulin secretion and enhanced lipogenesis and reduced lipolysis in adipose tissue (Sutton et al, 1980, Bines, 1982). However, in this trial consistently higher milk fat contents on the low concentrate to silage ratio diets were not found. This may have been attributable to the feeding of a complete diet, since high feeding frequencies are thought to result in a more constant and a lower decrease in pH (Kaufmann, 1976). However, it is unclear as to why a significant interaction between concentrate to silage ratio and trimming in relation to milk fat content should have occurred in period 1.

The higher milk protein contents found in the high concentrate group are in accordance with the results of Phipps et al (1984), who found significantly higher milk protein contents when cows were fed a ration containing 65% concentrate, as compared to one containing 50% concentrate.

The significantly higher milk protein yields found in the high concentrate group in periods 1 and 2 were due to the significantly higher milk yields and milk protein contents of the high concentrate group in these periods. Similarly, the the higher milk fat yields found on the high concentrate

treatments in periods 1 and 2, would have resulted from the significantly higher milk yields of those treatments in periods 1 and 2.

As in the previous trial, there was evidence that poor locomotion had a detrimental effect on production, and in particular on condition. The low condition scores associated with the high locomotion scores may have been due to the significantly ($p<0.001$) shorter times spent eating by the cows with poorer locomotion. However, although there were negative correlations between complete diet intake and locomotion score these were not significant. Therefore, lameness may have affected production in an additional way, for example, at the tissue level, through possible effects on factors related to the lipolysis or lipogenesis of adipose tissue, or through effects on the blood circulation of metabolites involved in milk production. Endotoxins associated with laminitis are known to affect blood circulation, although the extent to which they do so is little understood.

It is uncertain as to why there should be more significant correlations between locomotion score and various production parameters for treatments LCUT as compared to the other treatments. The difference may lie in the type and severity of clinical disease found in each treatment. Although the mean locomotion score for treatment LCUT was less that those of treatments HCT and HCUT, the proportion of clinical cases (0.20) which were scored as being 4 or more was nearly twice that of treatment HCT (0.12) and of treatment HCUT(0.09). Many of the clinical cases of lameness on treatment LCUT were attributable to double sole/sole separation type lesions. A more severe case of clinical lameness may have a proportionately greater effect on production through its possible effects at the tissue level than a less severe case.

SUMMARY

The effects of concentrate to silage ratio in a complete diet and Dutch hoof trimming were measured during weeks 3 to 26 of lactation on 48 autumn-calving cows. The four treatments were LCT (38:62, concentrate to silage ratio, hooves trimmed); LCUT (38:62, untrimmed); HCT (63:37, trimmed); HCUT (63:37, untrimmed). The cows on the low concentrate to silage ratio were fed ad libitum, and the high concentrate to silage ratio intake was adjusted so that CP and ME intakes were maintained at the same level for all treatments. Cows were scored for their locomotion weekly on a scale of 1 to 5 (high scores indicated poor locomotion), and hoof growth, wear and hardness measurements were recorded every eight weeks on the right hind outer claw. Hoof shape measurements were also recorded on the outer and inner claws of the right hind foot every eight weeks.

The high concentrate to silage ratio significantly increased locomotion score and trimming reduced locomotion score although not significantly, as compared to the low concentrate to silage ratio. Higher incidences of lameness were found both in the high concentrate to silage ratio treatments and in the untrimmed treatments. Similar numbers of clinical cases of solar problems and foot rot were found.

Trimming significantly increased hoof growth. Locomotion score was positively correlated with toe length and negatively correlated with toe angle and heel length.

The high concentrate to silage ratio significantly reduced the hardness of the abaxial wall and sole. Significant positive correlations between locomotion score and abaxial wall toe hardness, and significant negative correlations between height of outer heel and length of outer heel with heel bulb centre hardness were found.

Within treatments there were indications that poor locomotion and lameness were associated with losses in production. There was also an across treatment negative correlation between condition score and locomotion score.

CHAPTER 5

EXPERIMENT 4. The effect of lameness, nutrition and trimming on behaviour

INTRODUCTION

Production losses incurred as a result of a cow becoming lame are often ascribed to an increase in time spent lying with a concurrent reduction in time spent feeding. However, little research has been carried out to quantify this. A knowledge of whether lame cows adapt their eating behaviour to compensate for any reductions in feeding time would be useful for understanding the relationships between production and lameness.

Alterations in social behaviour, which may result from a deterioration in mobility, may also affect feeding and lying behaviour. In a group feeding situation access to a feeding place by a cow who is low in the dominance order may partly depend on her ability to avoid social interactions with more dominant cows. Therefore good locomotion would be an advantage under such circumstances.

These behaviour studies attempted to examine the relationships between losses in production and alterations in behaviour brought about by reduced mobility. Changes in social behaviour and their possible implications for feeding behaviour were also investigated. Thus, locomotion score was related both to time spent by the cows in various activities and to changes in social interactions.

MATERIALS AND METHODS

Three 24 hour observations were carried out in each of the three feeding trials, which are outlined below.

Experiment 1 (concentrate level trial)

Twelve heifers and 36 cows were allocated to two treatments:

7 kg concentrate/day fed at a flat rate (low)

11 kg concentrate/day fed at a flat rate (high).

the concentrate was fed from out-of-parlour-feeders and silage was ad libitum in a feed passage. The cows were housed in a kennel buiding and the cubicles were bedded with sawdust. The cows were locomotion scored weekly. The full details of this experiment are described in Chapter 2.

Experiment 2 (protein level, trimming trial)

Forty cows and 8 heifers were allocated to four treatments:

16% CP, feet trimmed (LT)

16% CP, feet untrimmed (LUT)

20% CP, feet trimmed (HT)

20% CP, feet untrimmed (HUT)

The diet was fed as a complete diet, and the amount offered to the cows on the high protein level was restricted to the DM intakes of the cows on the low protein level. The cows were housed in a cubicle building and the cubicles were bedded with sawdust. The cows were locomotion scored weekly. The full details of this experiment are described in Chapter 3.

Experiment 3 (concentrate to silage ratio, trimming trial)

Forty-eight cows were allocated to four treatments:

concentrate:silage ratio - 38:62, hooves trimmed (LCT)

concentrate:silage ratio - 38:62, hooves untrimmed (LCUT)

concentrate:silage ratio - 63:37, hooves trimmed (HCT)

concentrate:silage ratio - 63:37, hooves untrimmed (HCUT)

The cows were housed as in Experiment 2, and were locomotion scored weekly. The full details of this experiment are described in Chapter 4.

Lying, standing and feeding behaviour

Dates of observations for the first experiment (concentrate level trial) were 5 to 6 December, 1983, 30 to 31 January, 1984

and 19 to 20 March, 1984; for the second experiment (protein level trial) 3 to 4 December, 1984, 31 January to 1 February, 1985 and 18 to 19 March, 1985, and for the third experiment (concentrate to silage ratio) 5 to 6 December, 1985, 23 to 24 January, 1986 and 27 to 28 February, 1986. The cows were identified by their ear tag numbers and freeze brand numbers, and a team of observers recorded their behaviour manually at 10 minute intervals. Records of the following behaviour activities were made:

lying
standing in cubicle
standing in cubicle with forelegs in cubicle, hindlegs on concrete
standing on concrete (all four feet)
eating silage (Expt.1) or eating complete diet (Expts. 2 and 3).

The total time spent standing was calculated from the times spent standing in cubicle, standing with forelegs in cubicle, hindlegs on concrete, standing on concrete and eating silage or complete diet. The time spent occupying the cubicle was calculated from the times spent lying and standing in the cubicle.

Social behaviour

Dominance values were evaluated for the cows in each of the two feeding groups for each of the three experiments. Observations were made during 60 minute periods every day in the late morning or early afternoon to avoid the main feeding period, and therefore confusion between behaviour relating to competition and behaviour relating to dominance. The method devised by Beilharz and Mylrea (1963) and developed by Beilharz and Zeeb (1982) was used, as described below. The procedure involved the recording of all clear indications of dominance and both dominant and subordinate animals were identified. Such indications included one cow bunting another, or shaking her head at another with the result of movement away by the cow, or a cow standing her ground and forcing another to move around her. Doubtful interactions, for example, those where a movement may have been influenced by a competing motivation towards food, and interactions involving a cow showing signs of oestrus were ignored. Such observations were collected until every cow had been observed against at least 10 other cows. Every observation

even if it was a repeated observation of a previously recorded relationship was recorded.

Unclear relationships where X was sometimes dominant to Y, and sometimes submissive were also recorded. The proportions of observations in which X was dominant was credited as dominant to X, and a complementary portion as dominant to Y. (For example if X dominated Y 3 times, and Y dominated X once, X would receive 3/4 dominance and Y would receive 1/4 dominance for this relationship).

The dominance value of an animal X was calculated as the arc sin transform of the square root of the proportion of cows over which X was dominant, compared against all animals with which X had recorded relationships.

During the time when the dominance values were being assessed, the number of social interactions was recorded.

In the second and third experiments, whenever a cow became lame, or whenever her locomotion score was increased by 1.5 or more locomotion units, she was observed against at least four other cows with whom previous social interactions had been recorded.

Statistical analysis

The means calculated from the three 24 hour behaviour observations, carried out in each of the experiments, were used in the analysis of the results. For the first experiment the results were analysed as a randomised block design using the statistical package Genstat 5 Mark 4.03 (Lawes Agricultural Trust, 1980). For the second and third experiments, the results were analysed as 2x2 factorial designs using the Edex statistical package (Hunter, Patterson and Talbot, 1973, Edinburgh). A missing value was used in the second experiment as a cow died from a complicated mastitic condition. Minitab (Pennsylavania State University, 1980) was used to calculate correlation coefficients and regression coefficients between the various measurements.

RESULTS

Lying, standing and feeding behaviour

Means of the behaviour activities calculated from the three 24 hour (1440 minutes) observation periods, carried out in each of the three experiments are shown in Tables 5.10 to 5.12. The mean diurnal variations in the behaviour activities of each feeding group of the three experiments are shown in Figures 5.1 to 5.3.

In Experiment 1 the cows on the high concentrate group spent significantly ($p<0.05$) more time lying, and correspondingly less time standing, as compared to the cows in the low concentrate group. Differences in time spent eating silage were significant ($p<0.001$), with the cows on the low concentrate group spending more time eating.

In Experiment 2 the cows in the low protein group spent significantly ($p<0.05$) more time eating the complete diet than those in the high protein group. There were significant differences between the protein groups in time spent standing on concrete. The significant interaction between protein level and trimming treatment indicated that in the high protein group trimming decreased the time spent standing, whereas in the low protein group trimming increased the time spent standing. However, trimming by itself had no significant effects on any of the behavioural activities measured.

The cows on the low concentrate to silage ratio in Experiment 3 spent significantly less time lying ($p<0.05$) and occupying the cubicle ($p<0.01$) than those on the high concentrate to silage ratio, and there were corresponding differences between the groups in time spent standing on concrete and total time spent standing. The cows on the low concentrate to silage ratio spent significantly ($p<0.001$) more time eating the complete diet. No effects of trimming on any of the behavioural acitivities were found.

The rates of eating (minutes/kg DM), calculated from the mean times spent eating and mean silage and complete diet intakes for each dietary group, are shown in Table 5.13.

Correlation coefficients and regression equations between

Table 5.10 Behaviour activity means (minutes)@

Concentrate level trial (Experiment 1)

	Treatments		
	Low	High	SED
Lying	567	666	38.7*
Standing in cubicle	75	62	13.0NS
Cubicle occupation	642	728	41.2NS
Standing with forelegs in cubicle, hindlegs on concrete	191	147	27.7NS
Eating silage	283	218	11.1***
Standing on concrete (all four feet)	605	565	31.6NS
Standing (total)	873	774	38.2*

@ time spent for activity

Table 5.11 Behaviour activity means (minutes)$

Protein level trial (Experiment 2)

	Treatments						
	LT	LUT	HT	HUT	protein (P)	trim(T)	PxT
Lying	616	664	652	621	32.9NS	32.9NS	46.6NS
Standing in cubicle	69	102	103	107	24.1NS	24.1NS	34.0NS
Cubicle occupation	685	766	755	728	41.2NS	41.2NS	58.3NS
Standing with forelegs in cubicle, hindlegs on concrete	127	139	189	180	31.0NS	31.0NS	43.9NS
Eating complete diet	283a@	269ab	241b	254ab	10.8*	10.8NS	15.3NS
Standing on concrete (all four feet)	628a	535b	496b	532b	24.4**	24.4NS	34.6*
Standing (total)	824	776	788	819	36.3NS	36.3NS	53.9NS

$ time spent for activity

@ means with different subscripts are significantly different

Table 5.12 Behaviour activity means (minutes)$

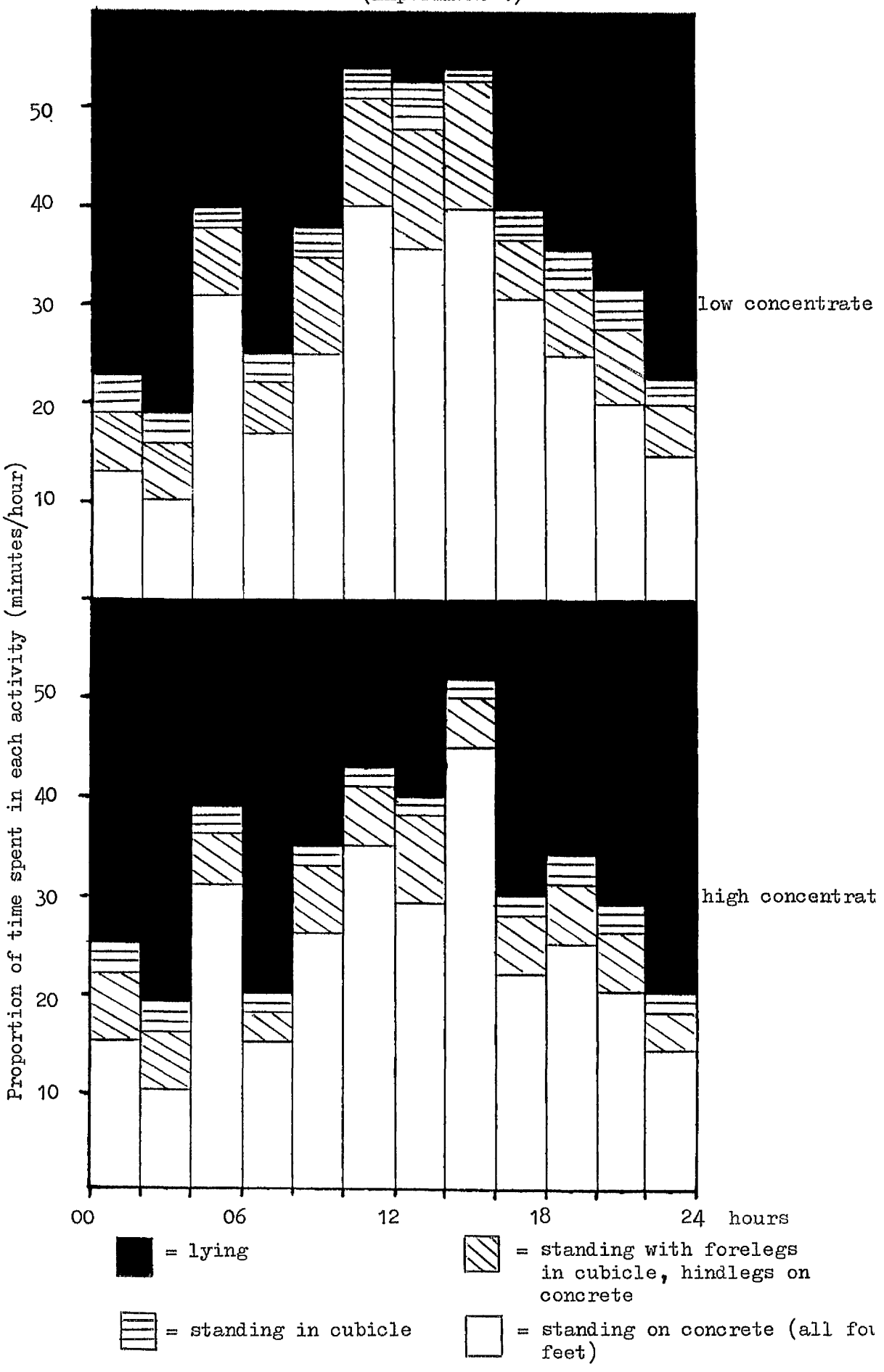
Concentrate to silage ratio trial (Experiment 3)

	Treatments						
	LCT	LCUT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Lying	552a@	596ab	687b	675b	40.3*	40.3NS	57.0NS
Standing in cubicle	136	99	147	133	21.3NS	21.3NS	30.2NS
Cubicle occupation	688a	695a	834b	808ab	47.2***	47.2NS	66.7NS
Standing with forelegs in cubicle, hindlegs on concrete	176	147	107	155	27.3NS	27.3NS	38.6NS
Eating complete diet	309a	333a	183b	192b	8.9***	8.9NS	12.6NS
Standing on concrete (all four feet)	563ab	598a	495b	479b	30.2**	30.2NS	42.7NS
Standing (total)	875a	844ab	749b	767ab	41.5*	41.5NS	58.7NS

$ time spent for activity

@ means with different subscripts are significantly different

Figure 5.1 Diurnal variation in behaviour
(Experiment 1)



(Experiment 2)

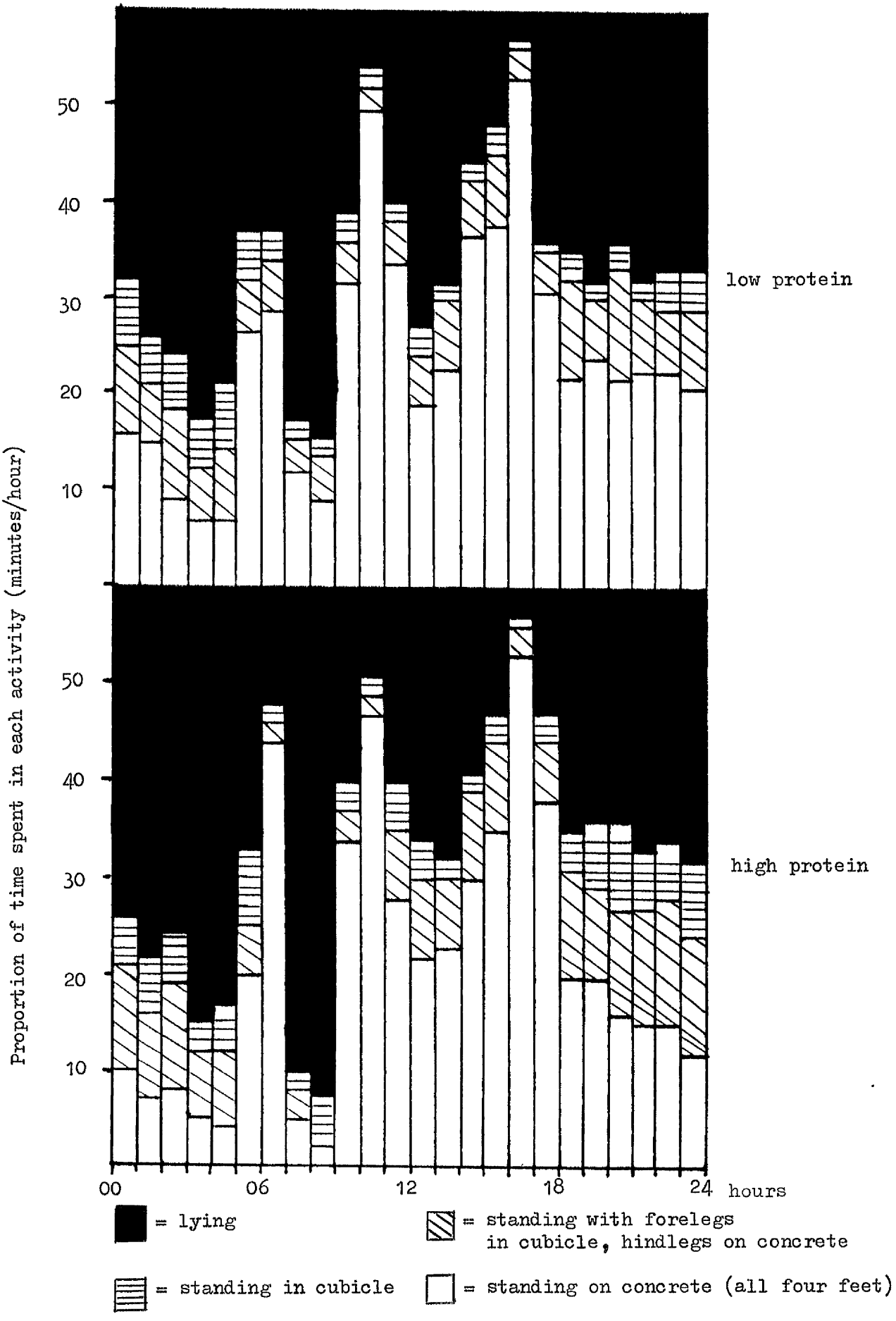


Figure 5.3 Diurnal variation in behaviour (Experiment 3)

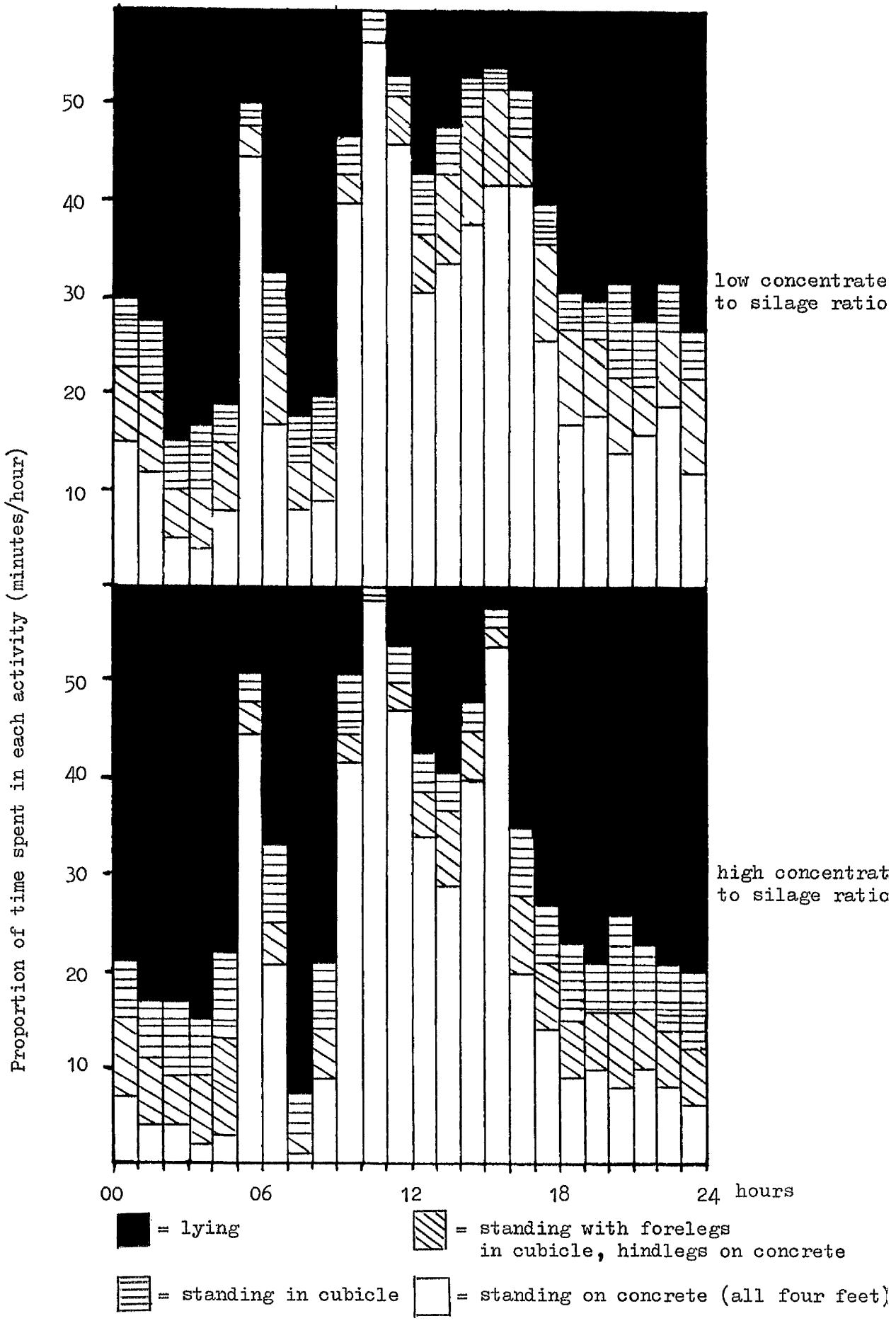


Table 5.13 Mean eating rates (minutes/kg DM) of silage and complete diet

Experiment 1 (concentrate level trial)

	Treatments	
	Low	High
Eating rate (min/kg DM)	35.4	32.5

Experiment 2 (protein level trial)

	Treatments			
	LT	LUT	HT	HUT
Eating rate (min/kg DM)	19.4	17.9	16.3	17.6

Experiment 3 (concentrate to silage ratio trial)

	Treatments			
	LCT	LCUT	HCT	HCUT
Eating rate (min/kg DM)	22.7	24.3	14.4	15.0

various behaviour traits and locomotion scores are shown in Tables 5.14 to 5.16. In all three experiments there were significant, negative correlations between time spent eating the silage, or complete diet, with locomotion score. However, the correlations between locomotion score and eating rate, indicated that as the cows became lame they spent less time eating 1 kg DM. The significant positive correlation between lying time and locomotion score in Experiment 2, and the significant positive correlations between time spent standing in the cubicles and time spent occupying the cubicles found in Experiments 2 and 3, indicated that as locomotion score increased, the time spent on softer surfaces ie. on sawdust bedded cubicles, increased. Correspondingly, there were negative correlations between time spent standing on concrete and locomotion score.

Social behaviour

Correlation coefficients and regression equations for number of social interactions with locomotion score are shown in Table 5.17, and correlations coefficients and regression equations for dominance value and the various behaviour activities are shown in Table 5.18. Correlation coefficients and regression equations between eating rate with dominance value and locomotion score are shown in Table 5.19.

In Experiment 1 no significant correlations between dominance value and the behaviour traits were found.

The significant ($p<0.05$) negative correlations between time spent eating the complete diet and dominance value found in the second and third experiments, indicated that time spent eating decreased with increasing dominance value. No signifiant correlations between dominance value and complete diet intake, as calculated from mean ME outputs per day for individual cows, were found. However, the significant correlation found between eating rate and dominance value in Experiment 2, indicated that less time was spent eating 1 kg DM as dominance value increased. In Experiment 2 there were significant ($p<0.05$) negative

Table 5.14 Correlation coefficients and regression equations for various behaviour activities (Y) @ on locomotion score (X)

Concentrate level trial (Experiment 1)

Lying (Y) on locomotion score (X)	r=-0.032NS	y=634-11(±51.1)X
Standing in cubicle (Y) on locomotion score (X)	r=+0.231NS	y=25+29(±17.9)X
Cubicle occupation (Y) on locomotion score (X)	r=+0.051NS	y=659+18(±51.5)X
Standing in cubicle with forelegs in cubicle, hindlegs on concrete (Y) on locomotion score (X)	r=+0.227NS	y=94+50(±31.6)X
Eating silage (Y) on locomotion score (X)	r=-0.333 (p<0.05)	y=322-48(±20.2)X
Standing on concrete (Y) on locomotion score (X)	r=-0.248NS	y=688-68(±39.2)X
Standing (total) (Y) on locomotion score (X)	r=-0.149NS	y=713-39(±38.6)X

@ time spent for activity

Table 5.15 Correlation coefficients and regression equations for various behaviour activities (Y) @ on locomotion score (X)

Protein level trial (Experiment 2)

Lying (Y) on locomotion score (X)	r=+0.297 (p<0.05)	y=514+75(±35.8)X
Standing in cubicle (Y) on locomotion score (X)	r=+0.371 (p<0.05)	y=-13+64(±24.0)X
Cubicle occupation (Y) on locomotion score (X)	r=+0.430 (p<0.01)	y=501+139(±43.5)X
Standing in cubicle with forelegs in cubicle, hindlegs on concrete (Y) on locomotion score (X)	r=+0.001 NS	y=160+0.20(±32.7)X
Eating complete diet (Y) on locomotion score (X)	r=-0.461 (p<0.01)	y=327-41(±11.8)X
Standing on concrete (Y) on locomotion score (X)	r=-0.554 (p<0.001)	y=756-126(±28.1)X
Standing (total) (Y) on locomotion score (X)	r=-0.480 (p<0.001)	y=1363-202(±55.1)X

@ time spent for activity

Table 5.16 Correlation coefficients and regression equations for various behaviour activities (Y) @ on locomotion score (X)

Concentrate to silage ratio trial (Experiment 3)

Lying (Y) on locomotion score (X)	r=+0.206	NS	y=508+67(±46.7)X
Standing in cubicle (Y) on locomotion score (X)	r=+0.396	(p<0.01)	y=1+72(±24.4)X
Cubicle occupation (Y) on locomotion score (X)	r=+0.380	(p<0.01)	y=509+138(±49.6)X
Standing in cubicle with forelegs in cubicle, hindlegs on concrete (Y) on locomotion score (X)	r=-0.075	NS	y=173-15(±29.6)X
Eating complete diet (Y) on locomotion score (X)	r=-0.562	(p<0.001)	y=422-94(±20.5)X
Standing on concrete (Y) on locomotion score (X)	r=-0.485	(p<0.001)	y=762-127(±33.9)X
Standing (total) (Y) on locomotion score (X)	r=-0.218	NS	y=936-71(±47.2)X

@ time spent for activity

Table 5.17 Significant correlation coefficients and regression equations for number of social interactions (Y) on locomotion score (X)

number of social interactions (Y) on locomotion score (X), Experiment 1 @	r=-0.414 (p<0.05)	y=30-6.5(±3.10)X
number of social interactions (Y) on locomotion score (X), Experiment 3 &	r=-0.430 (p<0.05)	y=41-10.4(±4.87)X
number of social interactions (Y) on locomotion score (X), Experiment 3 $	r=-0.390 (p<0.05)	y=36-7.4(±3.95)X

@ high concentrate group

& low concentrate to silage ratio group

$ high concentrate to silage ratio group

Table 5.18 Significant correlation coefficients and regression equations for behaviour activities (Y) on dominance value (X)

time spent eating complete diet (Y) on dominance value (X), Experiment 2 @	r=-0.424 (p<0.05)	y=325-1.4(±0.64)X
time spent eating complete diet (Y) on dominance value (X), Experiment 3 $	r=-0.454 (p<0.05)	y=218-0.8(±0.34)X
time spent standing on concrete (Y) on dominance value (X), Experiment 2 @	r=-0.422 (p<0.05)	y=733-4.0(±1.81)X
time spent standing on concrete (Y) on dominance value (X), Experiment 2 #	r=-0.417 (p<0.05)	y=619-2.7(±1.3)X
time spent occupying cubicle (Y) on dominance value (X), Experiment 2 #	r=+0.451 (p<0.05)	y=516+5.6(±2.4)X

@ low protein group

high protein group

$ high concentrate to silage ratio group

Table 5.19 Correlation coefficients and regression equations for eating rate (Y) on dominance value and locomotion score(X)

eating rate (min/kg DM) (Y) on locomotion score (X), Experiment 1	r=-0.109 NS	y=41.8-3.7(±4.97)X
eating rate (min/kg DM) (Y) on dominance value (X), Experiment 1	r=+0.034 NS	y=34.4+0.04(±0.16)X
eating rate (min/kg DM) (Y) on locomotion score (X), Experiment 2	r=-0.467 (p<0.001)	y=24.6-4.7(±1.13)X
eating rate (min/kg DM) (Y) on dominance value (X), Experiment 2	r=-0.577 (p<0.001)	y=23.6-0.2(±0.04)X
eating rate (min/kg DM) (Y) on locomotion score (X), Experiment 3	r=-0.462 (p<0.001)	y=18.6-4.0(±1.17)X
eating rate (min/kg DM) (Y) on dominance value (X), Experiment 3	r=-0.140 NS	y=12.5-0.03(±0.03)X

correlations between time spent standing on concrete and dominance value in both the low and high protein groups. The significant ($p<0.05$), positive correlation between time spent occupying the cubicle and dominance value found in the high protein group, showed that time spent in the cubicle, either lying or standing, increased with increasing dominance value.

Significant ($p<0.05$) negative correlations between the number of social interactions and locomotion score were found in the high concentrate group of Experiment 1 and both in the low and high concentrate to silage ratio groups of Experiment 3.

The results for the changes in social interactions with locomotion score are shown in Tables 5.20 and 5.21. In 90% (270 out of 300) interactions, the outcome of a social interaction of a cow with a mid (31-60) or high (61-90) dominance value after an increase in locomotion score, was the same as that previous to the change in locomotion. However, a cow with a low (0-30) dominance value became subordinate to a cow over whom previously she had been recorded as being dominant, in 86% (61 out of 71) interactions.

Table 5.20 Change in outcome of social interaction when locomotion score increased

Protein level trial (Experiment 2)

	Treatments	
	Low protein	High protein
No. of incidences where a cow of low dominance value (0-30) had an increase in locomotion score @	1 (4)$	3 (9)
No. of incidences where a cow became subordinate	4	8
No. of incidences where a previous outcome was maintained	-	1
No. of incidences where a cow became dominant	-	-
No. of incidences where a cow of mid (31-60) dominance value had an increase in locomotion score	17 (77)	19 (89)
No. of incidences where a cow became subordinate	4	4
No. of incidences where a previous outcome was maintained	70	81
No. of incidences where a cow became dominant	3	4
No. of incidences where a cow of high dominance value (61-90) had an increase in locomotion score	0 (0)	3 (13)
No. of incidences where a cow become subordinate	-	-
No. of incidences where a previous outcome was maintained	-	13
No. of incidences where a cow became dominant	-	-

$ the number in brackets is the number of social interactions recorded

@ An increase in locomotion score is an increase of 1.5 or more locomotion units or when a cow becomes lame. If there were 2 or more occasions when a cow increased her locomotion score then these were recorded as independant incidences.

Table 5.21 Change in outcome of social interaction when *locomotion score increased*

Concentrate to silage ratio trial (Experiment 3)

	Treatments	
	Low protein	High protein
No. of incidences where a cow of low dominance value (0-30) had an increase in locomotion score @	6 (40)$	4 (19)
No. of incidences where a cow became subordinate	34	15
No. of incidences where a previous outcome was maintained	5	3
No. of incidences where a cow became dominant	1	1
No. of incidences where a cow of mid (31-60) dominance value had an increase in locomotion score	4 (19)	16 (70)
No. of incidences where a cow became subordinate	3	8
No. of incidences where a previous outcome was maintained	16	60
No. of incidences where a cow became dominant	-	2
No. of incidences where a cow of high dominance value (61-90) had an increase in locomotion score	1 (6)	5 (26)
No. of incidences where a cow become subordinate	-	1
No. of incidences where a previous outcome was maintained	6	24
No. of incidences where a cow became dominant	-	1

$ the number in brackets is the number of social interactions recorded

@ An increase in locomotion score is an increase of 1.5 or more locomotion units or when a cow becomes lame. If there were 2 or more occasions when a cow increased her locomotion score then these were recorded as independant incidences.

DISCUSSION

Lying, standing and feeding behaviour

The cows on the high concentrate level in Experiment 1 spent significantly less time eating silage compared to the cows on the low concentrate level; this was reflected in their significantly lower silage intakes. The lower intakes of silage by the cows on the high concentrate level would have been due to the greater depressing effect of the high level of concentrate on silage intake. When forage is fed ad libitum additional concentrates generally reduce forage intake (Ostergaard, 1979). Concentrates affect the rate of forage digestion in the rumen, and the greater depression in the digestibility of cellulose with the the high level of concentrate, would have been caused by a reduction in numbers of cellulolytic and fibre digesting bacteria and an increase in number of lactic acid and propionic acid producing bacteria (Rook, 1975; Thomas and Rook, 1981). The significantly shorter time spent lying, by the cows on the low concentrate level, was probably a reflection of their significantly longer time spent in eating silage. Time spent eating silage is not necessarily indicative of silage intake, since intake is also dependant on the efficiency with which a cow uses her time at the feed passage (Friend et al, 1977). (Silage was offered ad libitum to both feeding groups).

In Experiment 2, the complete diet was fed ad libitum to the cows on the low protein level, and the DM offered on the high protein level was restricted to the DM intakes of the cows on the low protein level. The cows on the high protein level ate faster, and therefore spent less time eating 1 kg DM as compared to the cows on the low protein level, who spent significantly longer eating.

The significant interaction ($p<0.05$) between protein and trimming with respect to time spent standing on concrete, found in Experiment 2, is difficult to explain. There is no clear reason as to why trimming decreased the time spent standing of the cows on the high protein level, but increased standing time of the cows on the low protein level.

In the third experiment, the results showed that the cows on the high concentrate to silage ratio spent less time eating but ate more efficiently, as indicated by the eating rates. The lower eating times were probably a reflection of the lower amounts of the complete diet which were offered to the cows, and the faster eating rates were probably due to the complete diet of the high group being of a less fibrous nature, as compared to the complete diet fed to the low group. This is in accordance with the suggestion of Campling (1981) that feeds of a fibrous and bulky nature are eaten more slowly than those of a less fibrous nature.

As in Experiment 1, the greater time spent lying by the cows on the high concentrate to silage group may partly have been determined by the time spent eating the complete diet. However, before any firm conclusions can be reached, the strength of motivation of the cows for lying, standing or feeding need to be assessed and compared. For example, if the motivation for feeding is greater than the motivation for resting, then length of time spent feeding is more likely to determine length of time spent resting, rather than the other way around. All the environmental and social factors which may influence the motivation for feeding or for resting, for example, feeding space, degree of cubicle comfort, levels of aggression at the feed passage, hoof lameness etc. would then need to be considered in relation to motivation levels. The effect of lameness on feeding behaviour is discussed below.

At feed passages a competitive situation often exists, and an increase in aggression results from the concentration of cows in one place (Bouissou, 1981), and when the individual distance of a cow is violated. The space available for each cow at the feed passages in these trials was 0.7 metres, whereas the individual distance of a cow is thought to be between 1 and 2 metres (Sambraus, 1973). Mobility would be important in such a situation as it provides an alternative to competitive encounters (Fraser, 1980). Therefore, it might be expected that cows with poorer locomotion would avoid such situations and so spend less time eating, as was shown in all three trials by the significant,

negative correlations between locomotion score and eating time. Choice of foot placement for lame cows is also important, and Dewes (1978) has suggested that this choice may be denied if crowding is severe. Denial of such a choice may then deter lame cows from being in the vicinity of the feeding area. The decreased feeding time of the lame cows may also have been due to their increased lying time.

The positive correlations between time spent lying, time spent standing in the cubicle and time spent occupying the cubicle with locomotion score, in the second and third trials, were either a reflection of the the need of lame cows to alleviate the pain in their hooves by standing on soft surfaces, such as cubicles bedded with sawdust, or a reflection of the decreased eating times. A preference to avoid other cows, as discussed above, may also have explained these correlations, as might a combination of these factors.

Therefore, it is difficult to fully interpret the causal factors involved in the effects of higher locomotion scores in leading to lower eating times and longer lying times. Whether it is the alleviation of pressure in sore hooves with its resultant increases in lying time, the avoidance of competitive encounters, or the problems of foot placement associated with lameness, which is of most importance with respect to the relationships between lameness and eating time, and between lameness and lying time is difficult to assess, since the various motivations of the cow with respect to these factors and to feeding and lying itself are unknown.

Although time spent feeding by cows with poorer locomotion was less, complete diet intake was not significantly correlated with locomotion score. Lame cows compensated for their shorter periods spent feeding by eating more efficiently, as indicated by the significant correlations between eating rate and locomotion score in Experiments 2 and 3.

The decrease in eating time with increase in locomotion score would have contributed to the significant correlation found between time spent standing on concrete and locomotion score in

the second and third trials, since time spent eating the complete diet was a component of time spent standing on concrete.

Social behaviour

Friend and Polan (1974) have suggested that more submissive cows spend less time eating if the length of time during which the feed is available is limited. However, in these experiments time spent eating was significantly and negatively correlated with dominance value both in the low protein group in Experiment 2 ($p<0.05$), where the complete diet was available ad libitum, and in the high concentrate to silage ratio group in Experiment 3 ($p<0.05$), where the complete diet was restricted and usually eaten up after 6 hours. Therefore, the length of time during which the complete diet was available did not appear to influence the correlation between dominance value and time spent eating in these experiments. Other factors which may determine whether there is a correlation between dominance value and time spent eating include, levels of aggression within a group, stability of the dominance value and health problems, such as lameness. The correlation between a decrease in eating time with an increase in dominance value may only become significant if there are several cows with high dominance values, who also happen to have high locomotion scores, and if there are several cows with low dominance values who have low locomotion scores. If this situation was to occur, then the eating times of the cows with the high dominance values would be penalised by their inability to feed (feeding times are negatively correlated with locomotion scores), whereas the feeding times of the cows with the low dominance values would be relatively increased. However, it was not possible to make a valid statistical comparison between the number of cows who had high dominance values and high locomotion scores in the low protein and high concentrate to silage ratio groups with the other feeding groups, where non significant correlations between dominance value and time spent eating were found, since there were too few cows who both had high locomotion scores and high dominance values in the feeding groups. The effect of lameness on the relationship between dominance value and time spent eating requires further investigation.

In these trials there were no significant correlations

between dominance value and silage or complete diet intake, as measured by mean ME outputs per day of individual animals, and this may suggest that the effect of lameness on the relationship between dominance value and time spent eating is relatively unimportant with respect to production. The negative correlation between eating rate (min/kg DM) with dominance value, which indicated that the feeding efficiency of of those cows who spent less time feeding, ie. the cows with high dominance values, was greater than that of the cows with the low dominance values, probably accounted for the lack of a correlation between feed intake and dominance value. Likewise, several authors have found non significant correlations between milk yield and dominance value (Schein and Fohrman, 1955; Collis, 1976; Sambraus et al, 1979).

The significant correlations between time spent standing on concrete and dominance value, and the significant correlations between time spent occupying the cubicle and dominance value found in Experiment 2 may suggest that cows with higher dominance values have priority of access to cubicles, which provide a better environment for the hooves, over cows with lower dominance values. However, the locomotion scores of cows also seem to influence time spent in the cubicle, and these in addition to other motivational behavioural complexes, which are involved in the determination of competitive orders, and the differential learning experience of cows may influence priority of access as much as the learnt dominance relationships (Syme and Syme, 1979; Beilharz and Zeeb, 1982). Craig (1986) has also suggested that when resources become limited the majority may receive nearly equal awards, but those that are very low in status are affected severely. The lameness of cows with low dominance values may augment such a penalization. Therefore, before decisive conclusions about access to feeding space and cubicles in relation to lameness and dominance value can be reached, competitive orders and learning experience and their relevance to dominance values need to be assessed. However, it could be tentatively suggested that lame cows with high dominance values have an environmental advantage (in terms of access to cubicles) over lame cows with low dominance values.

Results from the second and third experiments indicated that

lameness affected cows with lower dominance values, with respect to their social behaviour, more than it affected cows with higher dominance values. Cows with low dominance values became subordinate to cows over whom previously they had been dominant if they became lame, whereas cows with mid or high dominance values maintained their position with respect to another cow when they became lame. This is in accordance with the results of Sambraus and Osterkorn (1974), who found that some cows maintained their high social rank long after their physical capacity to defend it had been lost. (Physical capacity would include factors such as hoof and leg health).

Cows with high locomotion scores are unsteady in their gait, and are therefore more likely to be affected by some of the physical factors involved in a social encounter than those cows who have a greater degree of mobility. Such physical factors include bunting and the awkward sideways or backwards movements, which are often invovled in the forced movement of a subordinate cow from the path of a dominant cow. Therefore, it is likely that a cow whose locomotion becomes impaired would learn to avoid such encounters. This was reflected in the results from all three trials, where significant, negative correlations between locomotion score and number of social interactions were found. Mobility would also be important in maintaining the individual space between cows, so that any aggressive encounters resulting from an incursion into another cow's space would be avoided. The avoidance of social interactions may also influence the time spent occupying the cubicle, since social interactions involving physical contact are at a minimum when a cow is standing or lying in the cubicle.

SUMMARY

Three 24 hour behaviour observations were carried out in each of the three feeding trials. Records of the following behavioural activities were made: time spent feeding, lying, standing in cubicle, standing with forelegs in cubicle and hindlegs on concrete, and standing on concrete (all four feet). The number of social interactions were recorded, and the dominance values were evaluated for the cows in each of the feeding groups.

Negative correlations between time spent eating the silage or complete diet with locomotion score, and positive correlations between lying time and locomotion score were found. Lame cows appeared to compensate in part for their shorter periods spent feeding by eating faster.

There were indications that the dominance value of a cow, in addition to locomotion score, may have influenced the time spent in the cubicle.

Negative correlations between the number of social interactions and locomotion score were found. When a cow with a low dominance value became lame or when her locomotion score was increased by 1.5 or more units, she was observed to become subordinate to cows over whom previously she had been dominant, in 86% of social interactions. However, the outcome of a social interaction of a cow with a mid or high dominance value after an increase in locomotion score, was the same as that previous to the change in locomotion, in 90% of social interactions.

GENERAL DISCUSSION

This series of experiments attempted to examine the relationships between nutrition, lameness, various hoof parameters and production. A further study investigated the effects of lameness on behaviour so that the interdependance between production and locomotion could be better understood.

The first experiment showed that high concentrate levels led to poorer locomotion and greater rates of clinical incidence. Two of the components of a high concentrate level - high CP intake and high concentrate to silage ratio - were then subsequently examined, (in Experiments 2 and 3, respectively), and both were implicated as being causal factors in lameness. Laminitis, through its effects on hoof formation and subsequent solar problems, was thought to be a major cause of lameness. In the third experiment laminitis also appeared to have aggravated the Dermatitis interdigitalis condition, present in many of the cows prior to their being on trial, which in turn led to a progressive horn erosion and underrunning. This aggravation may have explained why the high concentrate to silage ratio appeared to have a greater effect on lameness than the high CP intake. However, it is unclear whether the high protein level would also have led to such a progression, since there was little evidence of the existence of a Dermatitis interdigitalis condition in the cows at the start of Experiment 2.

It is unclear whether high CP intakes and high concentrate to silage ratios predispose to laminitis through similar mechanisms. However, in both cases endotoxins may be involved (Urmas, 1968; Andersson and Bergman, 1980). An examination of the series of events starting from when the nutrients thought to cause lameness are in the digestive tract, to the time when the horn formed as a result of the influence of such nutrients, reaches the surface of the hoof, would be useful. Until techniques have been developed to trace such pathways the role of nutrition in causing lameness will remain largely speculative. Since these experiments have established that certain nutritional factors are a major cause of lameness it would

seem expedient to develop such techniques.

Another nutritional factor which may be of importance in relation to lameness, is type of feedstuff fed. For example, different sources of protein or concentrate will have different amino acid compositions and crude fibre contents, which may make cows fed them more or less susceptible to lameness. Levels of energy intake may also merit examination. In high yielding cows the milk producing tissues are thought to compete with the horn building tissues for protein components (Hahn, 1979), and in Experiment 2 negative correlations between rate of hoof growth with milk yield and milk protein yield were found. Therefore, it would be interesting to investigate the effect of intakes of nutrients relative to requirements for milk production in relation to hoof growth.

The involvement of laminitis itself in causing lameness and its association with other digital diseases, merits the refinement and improvement of techniques, which would enable laminitis to be qualitatively and quantitatively assessed in the live animal. Results of the blood analyses from Experiment 2 showed no conclusive associations between levels of various blood parameters and lameness, and previous attempts at relating blood histamine levels and angiographic measurements to laminitis have also been unconvincing (Nilsson, 1963; Maclean, 1970; Greenough et al, 1981; Edwards, 1982). Although a diagnosis of laminitis based on the rotation of the pedal bone has proved to be reliable (Prentice, 1972; Greenough et al, 1981), the diagnosis can only be made at a post-mortem examination or from radiographs, both of which would be impracticable for an on-farm diagnosis. However, before appropriate tests can be devised the following question posed by Toussaint Raven (1973) would need to be answered: "Does laminitis have an acute onset and then become chronic or is a genuinely acute stage absent in several cases?" Until this question is resolved it is uncertain as to whether a test is needed to measure the gradual progression of the disease, or whether two tests are required (as would be the case if acute stages and chronic stages of the disease occured separately). This again indicates the need for an understanding of the pathways involved in laminitis.

The development of diagnostic tests would also be useful for

assessing the accuracy and potential use of the locomotion scoring system. The locomotion scoring system allows for a visual appraisal of laminitis through its reflection of some of the features of laminitis, for example, abduction and tenderness of the feet. It is also a useful indicator of the incidence (percentage of cows scoring 3 or more), severity (score) and duration (week by week score) of lameness in a herd, and of whether any preventative or curative measures need to be taken. However, it would be desirable to see how closely such features are symptomatic of what is occurring at the microscopic level, and also how locomotion scoring relates to other assessments of gait, for example the Selspot locomotion analysis system developed at the Scottish Farm Buildings Investigation Unit, Craibstone, Aberdeen, and the Institute for Research on Animal Diseases, Compton, Newbury.

Long, curly toes are thought to be evidence of chronic laminitis (Prentice, 1972; Livesey and Fleming, 1984), although it is also known that laminitis leads to a disruption of horn formation, which is likely to decrease rates of hoof growth and increase rates of hoof wear. The lower levels of net growth in Experiment 1, the lower rates of hoof growth in Experiment 2 and the higher rates of hoof wear in Experiment 3 may have been associated with a laminitic condition. However, it has also been suggested that the disruption of horn formation is relatively local, with stimulation of horn growth occurring in other parts of the hoof (Toussaint Raven, 1985). Relating toe length to chronic laminitis is problematic since overgrowths may be non-laminitic. For example, toe length may be caused by low rates of wear, as determined by amount of exercise and the abrasiveness of the concrete. Greenough et al (1981) have also suggested that excessive reliance should not be placed on any so-called characteristic shape of the laminitic hoof, since only in some cases is it flatter and wider than normal.

The use of trimming in preventing and curing lameness was apparent in these experiments. While low levels of concentrate and protein, and a low concentrate to silage ratio also improved locomotion and reduced clinical lameness, the adoption of such feeding regimes would penalize milk production and be difficult to incorporate into an existing management system. Trimming, however, would be easy to implement and is effective. Therefore, in herds where lameness is a problem, it would be advisable to

initially implement a trimming policy, and then if lameness were to persist to alter the feeding strategy.

Hoof shape greatly influences the likelihood of the development of hoof problems, since it is a major determinant of the action of the body weight on the solar corium. Solar problems were found to be the most frequently diagnosed cause of lameness in all three experiments. The importance of hoof shape and in particular toe length, was indicated by the effectiveness of trimming in reducing locomotion score, and the incidence of clinical lameness in Experiments 2 and 3.

Hoof hardness in addition to hoof shape is closely related to the susceptibility of a cow to become lame, and can be regarded as a measure of hoof quality. Whilst the sulphur amino acids are thought to directly affect hoof hardness, other nutritional factors, such as a high concentrate to silage ratio, through their predisposing to laminitis may also be of importance.

The hoof is not composed of homogenous tissue and thus the molecular structures contributing to the hardness of the hoof wall and the heel bulb may be as different as their mechanical functional requirements (Webb, 1984). Therefore, the various regions of the hoof are likely to respond in different ways to different nutritional and environmental conditions. For example, in Experiment 3, there was a significant treatment effect of the concentrate to silage ratio on the hardness of the wall and sole region, but not on the hardness of the heel bulb region. When considering treatment effects on hoof hardness it is important to recognise that while relatively hard hoof horn is beneficial in some regions, for example, in the mid sole and toe sole regions, where a hard sole would help to prevent solar penetrations, relatively softer horn is desirable in other regions. For example, soft horn is needed in the heel bulb centre region to allow the bulb to function as a cushioning, anti-concussive device.

From these trials it is apparent that hoof shape and hardness are major determinants of lameness, therefore, it might be useful to construct an index of hoof quality. Aspects such as hoof shape, hardness, elasticity and compressive strength could

be included in the index. The degree to which each of these factors influence a cows predisposition to lameness, within a particular set of environmental conditions, would need to be assessed. The factors could then be placed in order of importance and so an index constructed. The index could be used both in the establishment of criteria for the selection of breeding animals and in the assessment of an animals expected resistance to the development of hoof problems.

There have been many assertions, most of them undocumented, that lameness is the third most important disease syndrome in terms of economic loss, after mastitis and reproductive malfunction. It has frequently been supposed that production losses associated with lameness, are caused by decreases in feeding time, and therefore by decreases in food intake. In these trials, lameness had a detrimental effect on milk production in Experiment 3, and on condition score and liveweight in both Experiments 2 and 3, and significant negative correlations between feeding time and locomotion score were found. However, although negative correlations were found between feed intake and locomotion score, these were not significant. The lack of a significant result was attributed to the increase in feeding efficiency, as measured by eating rate, by the cows with poor locomotion. Since the correlations between feed intake and locomotion score were not significant, it was thought that some factor or factors, additional to changes in feed intake, might help to explain the relationship between lameness and production. Agents associated with lameness, which might in some way affect fat metabolism, and endotoxins associated with laminitis were suggested; these possibilities require further investigation.

Feed intakes in these experiments were calculated from estimated ME requirements for each cow on a weekly basis, since the cows were group fed. In future experiments examining the effects of lameness on production, it may be more expedient to use individual feed stands, since the model on which ME requirements is based, is not yet completely proven. Individual feeding would also remove the problem incurred in group feeding of increases in intake in non-lame cows being due to possible reductions in intake by lame cows.

Any reduction in health, by definition, means a reduction in well being (Duncan and Dawkins, 1983), since the physical state of the animal is impaired. It has been suggested that an animal is in a state of stress, if it is required to make abnormal or extreme adjustments in its behaviour or physiology, in order to cope with the adverse effects of its enviroment and management (Fraser, 1980). Lameness could be regarded as such an adverse effect. Therefore, the extent to which the welfare of the cow is compromised depends not only on the severity of the lameness with its concurrent alterations in physiology, but also on the extent to which the cow has to adjust her behaviour. However, whilst differences in adjustment can be used to compare different systems, these differences would give a relative rather than an absolute guide to the animal's welfare. The locomotion scoring system indicates to what extent the physical well being of the cow is impaired, therefore in one respect it is an indicator of welfare. However, mean locomotion scores obtained from cows under different management systems can not strictly be used to compare those systems, since different environments may require more or less adjustment in terms of behaviour for the same increase in locomotion score, depending on other factors such as access to feed etc.. For example, an increase in cubicle occupation by a cow who becomes lame may be dependant not only on the lameness itself, ie. on the need to alleviate the pain in her feet, but also on the need to avoid social encounters if the cow is low in the dominance order. If access to feed is limited, whether in terms of feeding space per cow or length of time during which the food is on offer, this need would be greater than in a non-competitive feeding situation, since any encounters would be more likely to involve an aggressive response (Metz and Mekking, 1978). Therefore, a proportionatley greater adjustment in terms of increased time spent in the cubicle would have to be made. Lameness could be regarded as more of a welfare problem in a management system where a competitive feeding situation exists, as compared to one where there is a non-competitive feeding situation. Therefore, if the amount of adjustment ie. the increase in time spent say lying for each increase in locomotion score, is compared for each different system, then this may give some indication of the extent to which the physical well being is impaired under each system. However, a more accurate comparison could be reached if the relative importance of each type of

behaviour for which adjustments were made, could be assessed. For example, is an adjustment in lying behaviour more indicative of a welfare problem than an adjustment in feeding behaviour?

These experiments attempted to identify some of the management factors which predispose to lameness. The results indicated that whilst trimming may to some extent alleviate hoof problems resulting from high input feeding systems, the levels of lameness incurred may still require adjustments in behaviour and losses in condition, which in some circumstances may constitute a welfare problem.

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APPENDIX 1 TYPES OF LAMENESS CAUSED BY HOOF PROBLEMS

The nomenclatura and definitions of diseases of the hoof as agreed upon at Skara in 1978 and oultined by Weaver et al (1981).

Pododermatitis aseptica diffusa (laminitis): a diffuse acute, sub-acute or chronic aseptic pododermatitis usually involving several feet. In chronic cases there are no generalized symptoms, but lameness and rigidity may persist. The shape of the claw may be altered ana the wall may be grooved. There is often discolouration of the solar horn.

Pododermatitis circumscripta (solar bruising/ulcer): a reaction of the pododerm often characterised by erosion of the horn. The severity of the lesion ranges from a bruising in the horn to an ulcer of the pododerm. The ulcer is usually located in the axial part of the sole-bulb junction in the outer hind claw. The disease is often seen in combination with mechanical or metabolic stress. A localized ischaemia of the corium, which follows the development of abnormal horn in the lateral hind claw, may lead to contusion and an open ulcer.

Dermatitis interdigitalis: an inflammation of the interdigital skin without extension to the deeper tissues. Bacteroides nodosus has been isolated ftom superficial lesions of the inter-digital skin. There may be separation of the horn on the inner aspects of the bulbs and entry of dirt between the horn and underlying dermis, with a resultant proliferative reaction. If under-running does not progress, the lesion becomes chronic and there is little lameness. Complications as a result of under-running of horn include solar ulcers and irregular loss of tissue from the horny capsule.

Erosio ungulae (together with Dermatitis interdigitalis is commonly known as foot-rot): an irregular loss of solar or bulbar surfaces of the horny capsule. V-shaped clefts in the horn of the bulb are characteristic and the condition is often followed by horn overgrowth of poor quality. Raven and Cornelisse (1971) drew a direct relationship between Interdigital dermatitis and erosions occurring in the bulbs.

Phlegmona intergigitalis (foul-of-the-foot): an inflammtion of the inter-digital skin and the underlying tissues, originating in the dermis and characterised by necrosis and fissures in the skin and swelling. Fusobacterium necrophorum has been recovered consistently from the lesion.

Pododermatitis septica (solar penetration): a diffuse or localized septic inflammation of the pododerm, following penetration of the horny capsule. The condition usually follows trauma involving a foriegn body or excessive abrasion.

Hyperplasia interdigitalis (inter-digital growth): a proliferative reaction of the inter-digital skin and, or subcutaneous tissue, especially dorsally. The process is chronic and caused by contact irritation.

Appendix 2 Dutch trimming method

From the age of 18 to 24 months differences between the outer and inner claws are perceptible. The lateral claw is larger than the inner claw and the sole is flatter. The heel bulb of the outer claw is also wider and the heel is higher. The higher heel results in a measurable increase in weight bearing by the outer claw, which causes discomfort and contusion of the corium (Toussaint- Raven, 1973). One of the main objectives of functional trimming is to correct this incorrect loading. This is achieved by shortening overgrown walls to attain a better division of weight within the claws with the weight being borne more anteriorly and abaxially, and by trimming the outer and inner claws to the same height and length so that there is a better division of weight over both claws. By the removal of loose horn and the thinning of horny rims, pressure points are avoided and penetration of dirt is prevented. Trimming a plane bearing surface underneath the claw also ensures a stable supporting surface on hard ground (Toussaint-Raven, 1985).

The method described below is adapted from the guide on foot care published by the Department of Large-Animal Surgery, Utrecht University and the Agricultural Education Division, Ministry of Agriculture and Fisheries, The Netherlands (1982).

Functional trimming

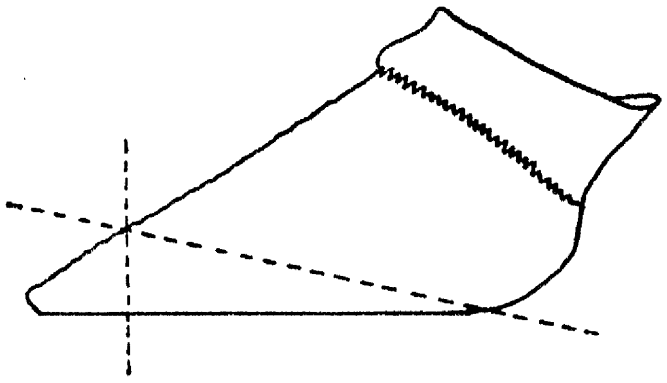
1. Restrain the cow and clean the hind hooves.
2. Measure the length of the inner claw and if necessary clip the claw to about 7 cm. (The length will slightly vary depending on the size of the cow).
3. Cut a plane bearing surface (a surface perpendicular to the axis of the shin bone) underneath this claw, see diagram.
4. Clip the outer claw to the same length as the inner claw, and trim the heel to the same height as the inner claw, see diagram.
5. If necessary, shape the axial borders of the soles, so that the hooves becomes self-draining.

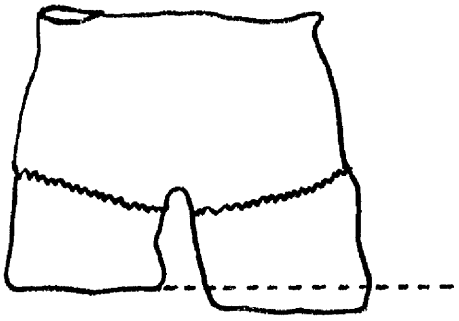
Curative trimming

6. Trim away any loose horn and trim down horny borders.
7. Follow any cracks or fissures out.

Claw problems usually occur in the hind hooves, however trimming of the front hooves is desirable if they become overgrown or "cork- screw like."

Dutch trimming method





---------- trimming lines

Appendix 3 Initial Animal Production Data (14 day values)

	Treatments			
	Low		High	
	cows	heifers	cows	heifers
Milk yields (kg/day)	25.1	16.9	25.1	16.9
Liveweights (kg)	605	508	608	505
Condition scores	2.3	2.2	2.3	2.3
Parity	3.5	1.0	3.5	1.0

APPENDIX 4

Method used to calculate individual silage DM intakes from weekly group silage intakes

1. Calculate the average ME ouptut per day for each indidual cow for the week in question using milk yield, milk composition, liveweight and liveweight change and the equations describea in MAFF (1975).

2. Add up the 24 average ME outputs for each individual cow in the group to get the mean ME ouput for the group for that week. Subtract from this the energy derived from the actual mean daily concentrates consumed in that week. This will give the energy contribution from the average amount of silage eaten for that week. Divide this by the ME content of the silage, which will give the theoretical mean silage DM intake for the group of 24 cows for that week.

3. Divide the mean actual quantity of silage DM eaten that week for the group by the theoretical amount eaten from 2 above. This will give the correction factor.

4. Calculate all 24 cows' individual theoretical silage DM intakes and correct these using the correction factor from 3 above. This will ensure that the mean of the 24 individual silage intakes corresponds exactly with the actual mean group intake for that week, measured from weights given and refused.

APPENDIX 5

Equations used to predict the Metabolisable Energy (MJ/kg DM) content of the feeds

$$\text{Silage ME} = \frac{(\text{in vitro OMD (\%)} \times 0.907 + 6.03) \times \text{OM (g/kg)} \times 0.16}{1000}$$

$$\text{Concentrate ME} = \frac{(\text{in vitro OMD (\%)} \times 1.207 - 10.21) \times \text{OM (g/kg)} \times 0.16}{1000}$$

Appendix 6 Locomotion scoring system

Explanation of terms used in the locomotion scoring system

1) Degree of abduction: tendency to rotate the hind or fore limb outwards, and hock inwards.

2) Degree of adduction: tendency to rotate the hind or fore limb inwards.

3) Eveness of gait: extent to which the length of stride and the height to which the moving limb is raised, is the same for both right and left sides of the cow.

4) Tenderness of feet: as judged by how willingly and firmly the feet are placed on the concrete.

5) Normality of behaviour pattern: for example, ease in rising, willingness to walk, degree to which interactions with other cows are avoided and ease of feeding.

6) Extent of tracking up: how close the hind foot is placed in the imprint of the fore foot.

The locomotion scoring system

Score 1.0: 75% plus tracking up
minimal abduction/adduction
no uneveness
no tenderness

Score 1.5: maybe less than 75% tracking up
slight abduction/adduction
no uneveness
no tenderness

Score 2.0 less than 75% tracking up
abduction/adduction present
uneven gait
maybe tender, possibly with arching of the back and downward extension of the neck and head

Score 2.5: less than 75% tracking up
abduction/adduction present
uneven gait
tenderness

Score 3.0: slight lameness, not affecting normal behaviour pattern

Score 3.5: lameness obvious, not affecting normal behaviour pattern difficulty in turning

Score 4.0: lameness very obvious, affecting normal behaviour pattern difficulty in turning

Score 4.5: lameness affecting normal behaviour pattern considerably unwilling to rise

Score 5.0: severely lame, adverse effects on behaviour and condition extreme difficulty in rising

Appendix 7

Initial Animal Production Data (14 day values)

	Treatments							
	LT		LUT		HT		HUT	
	Cows	Heifers	Cows	Heifers	Cows	Heifers	Cows	Heifers
Milk yields (kg/day)	25.2	16.1	26.8	14.5	25.2	15.6	25.4	16.2
Liveweights (kg)	571	463	572	455	571	472	566	469
Parity	3.2	1.0	3.3	1.0	3.2	1.0	3.2	1.0

Appendix 8

Hoof shape means

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Initial							
Length of toe (outer) (cm)	7.98a@	9.45b	8.08a	9.31b	0.19NS	0.19***	0.27NS
Length of toe (inner) (cm)	7.68a	8.86b	7.84a	8.56b	0.15NS	0.15***	0.22NS
Length of heel (outer) (cm)	3.70	3.99	3.87	3.93	0.16NS	0.16NS	0.23NS
Length of heel (inner) (cm)	3.44a	3.89b	3.63ab	3.77ab	0.14NS	0.14*	0.19NS
Final							
Length of toe (outer) (cm)	9.08	9.34	9.66	9.80	0.33NS	0.33NS	0.47NS
Length of toe (inner) (cm)	8.43	8.35	8.32	8.57	0.14NS	0.14NS	0.20NS
Length of heel (outer) (cm)	3.69a	3.93ab	4.08b	3.73ab	0.13NS	0.13NS	0.18*
Length of heel (inner) (cm)	3.59	3.83	3.78	3.62	0.12NS	0.12NS	0.17NS

@ means with different subscripts are significantly different

Appendix 9 Hoof hardness means (Shore A units, scale 0 to 100)

	Treatments				SED		
	LT	LUT	HT	HUT	protein(P)	trim(T)	PxT
Initial							
Abaxial wall toe	85.7	83.9	90.4	83.8	2.79NS	2.79NS	3.94NS
Abaxial wall mid	87.0	80.7	83.3	81.3	2.74NS	2.74NS	3.88NS
Sole toe	80.9	79.0	77.3	73.6	3.18NS	3.18NS	4.49NS
Sole mid	75.8a@	63.7b	69.8ab	66.7b	3.05NS	3.05*	4.31
Heel bulb centre	36.2	42.2	37.0	37.7	2.99NS	2.99NS	4.23NS
Final							
Abaxial wall toe	86.3	84.0	82.8	82.9	1.65NS	1.65NS	2.33NS
Abaxial wall mid	84.6ab	82.6a	83.9a	88.3b	1.50NS	1.50NS	2.12*
Sole toe	82.7	81.8	84.4	80.6	1.57NS	1.57NS	2.21NS
Sole mid	76.6a	74.4ab	72.7ab	71.5b	1.65*	1.65NS	2.34NS
Heel bulb centre	37.1	37.8	38.0	40.3	2.86NS	2.86NS	4.04NS

@ means with different subscripts are significantly different ($p<0.05$)

Appendix 10 Initial Animal Production Data (14 day values)

	Treatments			
	LCT	LCUT	HCT	HCUT
Milk yields (kg/day)	23.0	23.0	22.9	23.4
Liveweights (kg)	569	564	569	566
Parity	3.5	3.6	4.4@	3.6

@ this high parity was caused by one cow being in her seventh lactation and one cow being in her ninth lactation.

Appendix 11

Hoof shape means

	Treatments				SED	
	LCT	LCT	HCT	HCUT	C:S ratio (R)	trim(T)	RxT
Initial							
Length of toe (outer) (cm)	8.20a@	9.38b	8.28a	9.63b	0.18NS	0.18***	0.26NS
Length of toe (inner) (cm)	7.98a	8.51b	8.15ab	8.42ab	0.18NS	0.18*	0.26NS
Length of heel (outer) (cm)	3.63	3.56	3.69	3.68	0.14NS	0.14NS	0.20NS
Length of heel (inner) (cm)	3.55	3.38	3.53	3.51	0.12NS	0.12NS	0.16NS
Final							
Length of toe (outer) (cm)	9.17	9.58	9.08	9.48	0.32NS	0.32NS	0.46NS
Length of toe (inner) (cm)	8.58ab	8.81ab	8.53a	9.01b	0.16NS	0.16*	0.23NS
Length of heel (outer) (cm)	3.35	3.43	3.62	3.34	0.17NS	0.17NS	0.24NS
Length of heel (inner) (cm)	3.27	3.26	3.41	3.07	0.16NS	0.16NS	0.22NS

@ means with different subscripts are significantly different

Appendix 12 Hoof hardness means (Shore A units, scale 0 to 100)

	Treatments				SED		
	LCT	LCUT	HCT	HCUT	C:S ratio(R)	trim(T)	RxT
Initial							
Abaxial wall toe	85.8	85.3	83.7	85.1	1.39NS	1.39NS	1.97NS
Abaxial wall mid	84.2	83.8	84.3	82.4	1.42NS	1.42NS	2.00NS
Sole toe	83.5a@	76.9b	79.3ab	78.8b	1.57NS	1.57**	2.22NS
Sole mid	74.4	74.2	68.2	70.9	2.37NS	2.37NS	3.35NS
Heel bulb centre	41.0	45.3	40.2	43.3	1.98NS	1.98NS	2.80NS
Final							
Abaxial wall toe	85.1	85.5	83.5	84.8	1.37NS	1.37NS	1.94NS
Abaxial wall mid	82.3	84.5	83.3	85.3	1.45NS	1.45NS	2.05NS
Sole toe	83.9ab	85.9a	81.1ab	79.8b	1.72*	1.72NS	2.43NS
Sole mid	80.3a	81.3a	74.3b	75.5b	1.55***	1.55NS	2.19NS
Heel bulb centre	35.7	36.8	36.8	30.8	3.23NS	3.23NS	4.57NS

@ means with different subscripts are significantly different ($p<0.05$)